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THE NATURE OF SYMPTOMS

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PATHOLOGICAL ANATOMY VERSUS PATHOLOGIC PHYSIOLOGY

Present-day medicine owes the superiority which it possesses over the medicine of the centuries past particularly to a number of developments which began in the middle of the last century and which are still going on.

It is impossible for a subject so many sided as medicine to unfold logically. Its progress must follow the direction of those who, at the time, are able to impress the leaders in medical thought most; so an orderly, logical unfolding is not to be expected. The transference of certain disease syndromes to the organs or systems which give origin to them took place early and was the cause which inspired the study of anatomy. It was natural to want to know the difference between a normal organ and the same organ when it was the seat of disease, hence the development of gross pathology.

After the use of the microscope became general it was utilized in the study of both normal and pathologic tissues and histology and microscopic pathology were developed. These structural phenomena seemed so important and so fundamental to the understanding of diseases, and, at the same time, their study became so fascinating, that they early dominated the whole subject of medicine. Clinicians seemed to forget for the time being that their major problem in the practice of medicine was to understand and correct symptoms which are for the most part due to deranged function. The tendency to get farther away from the study of deranged function was increased by the next great development in medicine,—that of the bacterial cause of disease.

The study of the structural changes, particularly those caused by microorganisms and the desire to know every minute phase of bacterial growth and development put microscopic medicine to the fore, and completed the establishment of the hierarchy of structural pathology. Thus the study of structure developed much faster than the study of function. It carried with it elements of accuracy hitherto unknown to clinicians, and so was greatly appreciated and readily accepted. This accuracy was such that it could be duplicated in the study of symptoms neither by the knowledge then at hand, nor by the knowledge since developed; for the study of function is surrounded with much greater difficulties than the study of structure.

In studying function one is confronted by many variables, each of which may modify symptoms of disease. Symptoms may be thus modified by inherited characteristics, physico-chemical differences, endocrine predominances and different states of nervous stability or instability that are found in different patients, or in the same patient at different times. All of this again may be influenced by stimuli which arise either in the physical, emotional or psychical domain. When we realize that every disease process has its psychical side and its basis for emotional upets as well as its physical aspects, we can see how the study of function is beset with great difficulties, and how natural it was for its development to be one of the late accomplishments of medicine.

Now that the study of structural pathology has advanced to a fairly satisfactory state, the development of functional pathology, aided by the methods developed and the facts established, should go on much more

rapidly than has been heretofore possible. It is the particular phase of medicine above all others which, at the present time, is demanding our interest and intelligent cosideration.

The one significant fact about the physiologic aspects of medicine, as contrasted with its anatomical or structural aspects, is that in its study our minds are directed largely to the study of the patient who has the disease; to him, too, in both his physical and psychical spheres for disease affects and is modified by both spheres.

Pathologic changes in tissues and organs cause little concern to the patient who harbors them until they produce departures from the normal function in tissues and organs. These at once assume the importance of symptoms of disease, and effect real and definite changes in the individual's feeling of well being or in his outlook for future enjoyment and accomplishment.

The clinician must ever struggle to understand and alleviate the symptoms which he encounters in his patients. It matters not whether some structural pathology underlies them or not. If function is disturbed, it is the duty of the physician to find out why, in order that he may guard the best interests of the patient.

We shall now attempt to further elaborate this subject by analyzing a few concrete examples, hoping thereby to stimulate thought and further discussion.

IMPORTANCE OF ETIOLOGIC STUDY OF SYMPTOMS

In the fall of 1908, when palpating the chest of a patient suffering from active tuberculosis, I found that certain skeletal muscles were in a state of spasm. I considered this as probably due to the same mechanism that produces spasm of the abdominal muscles in appendicitis, gall-bladder disease and ulcer of the stomach. But when I endeavored to find out what that mechanism might be, to my great surprise I learned that the explanation of it was not only not common knowledge, but that it was understood by very few.

I then set about to find out how pulmonary inflammation could produce spasm of the muscles of the shoulder girdle. This led me into the study of the nerves of the lung, which, belonging to the vegetative system, had at that time received but scant attention at the hands of physiologists. In fact, physiologists had not yet worked out the reflex paths from the lung. Serious study of the visceral nerves was just beginning to be prosecuted by a few men. Langley, Anderson, Gaskell and Cannon had been experimenting on animals and were able to give account of certain experimental phases of the subject. Eppinger and Hess soon published their conception of Vagotonia; and Higler and Muller followed with a more general description of the clinical phases of visceral neurology. Since then the subject has been occupying an increasing amount of space in literature, but is still far from receiving the consideration that its importance demands.

I found that it was not only this new sign in pulmonary tuberculosis that was not understood, but symptoms in general were simply accepted as entities, without an adequate conception of the reason for their existence.

When I had obtained only a meager conception of visceral neurology, I saw how essential its development was to the understanding of clinical phenomena. I recognized that most of the symptoms of tuberculosis could be expressed in terms of disturbance in the vegetative nervous system. I then saw that the same was true of symptoms of other diseases. It seemed clear to me that the symptoms of tuberculosis were caused in one of three ways, and that all of the twenty or thirty common symptoms which accompany pulmonary tuberculosis could have but three etiologic factors; (1) the toxins of the disease; (2) stimuli arising in the inflamed lung and expressing themselves in reflex effects in other structures; and (3) effects produced locally by injury caused by the pathologic process.

It is evident that symptoms falling in groups one and two are definitely caused by disturbances in physiology produced for the most part at some distance from the seat of the disease, and that even those of group three are partly of physiologic origin. The increased permeability of blood vessels which permits of the passage of blood through their walls is a disturbance of normal physiologic function; the increased secretion which results from the increased activity of the mucous glands is caused through nerve stimulation; the pain and muscle tension which accompany pleurisy are evidence of nerve irritation, the seat of stimulation being transferred to the parietal pleura; and many of the symptoms of the so-called "cold" are of physiologic nature. This leaves the rupture of blood vessels; the secretion caused by necrotic foci which carries the bacilli; and the effusion in the pleura or the proliferation resulting from the pleural inflammation only as being distinctly of structural nature.

It is difficult to estimate the total number of symptoms that can be produced by a pul-

monary tuberculosis. There may be (1) so many as there are systems affected by the toxins; (2) plus the number expressed in systems and organs which may be brought into reflex connection with the lung through neurons which mediate with the afferent neurons which carry impulses from the lung; and (3) plus those produced locally by the inflammatory reaction. I have personally described some forty reflexes alone, which must be augmented by symptoms of toxic and local origin to make up the total number.

The chief fact that has been impressed upon me by this study is the unity of the human body, and the manner in which activity in one organ or system is correlated through nerves and chemical substances with effects in other organs or systems. It shows the futility of attempting to divide the study and practice of medicine into specialites of this and that organ, or this and that system; for diseases do not limit themselves in that way. If there is any illness in the body anywhere, the whole individual is sick, the only difference is in degree.

There is an emotional factor in every disease, which may cause distinct symptoms of its own through the thalamic centers. So simple a thing as a burned finger may affect every system of the body through the ramification of the sympathetic nerves which in the presence of pain receive stimulation through the thalamis centers. The same is true of tonsilitis, pneumonia, pyetitis, septicemia, or any other disease accompanied by pain, toxemia, or emotional stress; for toxemia and emotional stress, like pain, produce widespread effects througs central stimulation of the sympathetico-adrenal system. The whole body is made sick by pain and toxemia and emotions, but in some instances, as in the burned finger, the effects are so slight that very little or no recognizable disturbed function results but let the lesion be more severe and even a burned finger may at least produce recognizable vasomotor phenomena and inhibitory effects upon appetite and digestion.

As stated above, symptoms of disease are for the most part disturbances in function. Function may be so simple a thing as the taking up or giving off of salts or nutritive substances by the cells; but such is not a comprehensive statement of the function of a complex and complicated organ or system in the body or of the body itself. Function in this sense means activity in cells, organs or systems by which their normal physiologic purpose of action and correlation of action is expressed for the good of the individual.

Disturbed function then, as we recognize it as an expression or symptom of disease, means a departure in the normal purposeful working of some organ or system, or all organs and systems constituting the body. There is an instability or even a more complete disruption of normal activity brought about in the physiologic functions of the body. This disturbed function may be due to a change in activity in any portion of the neurocellular mechanism which is responsible for normal action. The collodial make-up of the cell itself; the electrolytes which are necessary to its function; chemical substances such as hormones, toxins and so forth, which reach it through the circulation, or the nerves which transmit stimuli to it; one or all may be at fault.

SYMPTOMS AND THE VEGETATIVE NERVOUS SYSTEM

I have learned to think of symptoms of disease in terms of the vegetative nervous system which supplies the structures which show faulty action. This has to recommend it the fact that all structures of the body are supplied by vegetative fibers belonging to the sympathetic system and many of them have parasympathetic innervation as well. Stimulation or inhibition in these systems means a fairly definite action. Then, too, these two systems of vegetative neurons have each their particular function to perform for the organism. The parasympathetic system is the one that presides over the digestion and assimilation of food and the maintenance of the human body as a working machine; the sympathetics, on the contrary, comprise those neuorns which protect the individual against enemies, prepare him for fight or flight, and help him to make a supreme struggle in the face of danger. The parasympathetics build up the organism; hence are anabolic in action; the sympathetics tear down, hence are catabolic.

In case we have a predominantly sympathetic action in the body, some of the following symptoms will be present; erection of the hairs, sweating, widening of the pupils, salivary inhibition, relaxation of the bronchi, increased pulse rate, elevation of blood pressure, increase of sugar in the blood, and a decrease in the motor and secretory functions of the gastrointestinal tract. A predominantly parasympathetic action is responsible for contraction of the pupil, increased secretion of the glands of the nasal and oral mucous membranes, constriction of the bronchi, slowing of pulse rate, lowering of blood pressure, storage of sugar in the liver, and an increasing of all activity,

whether secretory or motor, throughout the gastrointestinal canal.

By knowing these various effects one may readily classify disturbed function in organs which have double innervation according to the expression of activity of one or the other system of vegetative nerves without necessarily implying that the cause is in the nerve itself; for it may be in any component of the neurocellular mechanism. The effect, however, is more or less distinctly a hyper- or hypofunction, and so may be thought of in terms of the vegetative nerves supplying the structure.

Thus, increase in the number of heart beats means a sympathetic effect, while a slowing of the heart is a parasympathetic effect. Dilatation of the pupil is a sympathetic effect: constriction, a parasympathetic. Asthma consists of contraction of the bronchial musculature and an increase in the secretory activity of the bronchial glands, together with an exudation in the tissues which may be considered as a part of an increased vascular permeability, all of which is a part of a parasympathetic picture; and, so on.

ANALYSIS OF SYMPTOMS APPEARING WITH CHANGES IN BLOOD SUGAR LEVEL

Now, as illustrating the value of such a mode of analysis, let us take, as an example, a condition of equilibrium maintained in the body such as the sugar level of the blood.

The sugar of the blood is maintained at a fairly constant level under many and varying conditions and circumstances of health. When large quantities of sugar—too large for immediate utilization—are consumed, the excess is stored in the liver in the form of glycogen, to be given out in the natural course of events, as needed by the muscles; or as artificially called for after insulin has depleted the blood supply, as is seen in experimental hypoglycemia.

By studying the effects of insulin upon this store we can see a delicate physiologic balance. When insulin is given in such quantities as to reduce the glucose below 70 mgs. per 100 c.c. of blood, the hypoglycemic state may supervene. This state is met by a defense reaction on the part of the body brought about by the sympathetic nerves and the adrenal bodies coming to the rescue and forcing sugar out of its storehouse into the circulation. The rapidity of the pulse, the dilatation of the pupils, and the profuse sweating are further evidence of increased sympathetic action. If the sympathetico-adrenal system fails to restore the necessary amount of sugar to the blood and the amount of blood sugar continues falling, convulsions may appear. Convulsions stimulate still further the sympathetico-adrenal system and if the liver still contains an ample store of glycogen, the reaction may be able to force sufficient glucose into the blood stream to relieve the hypoglycemic state, and with it the convulsions; but if not, death may ensue.

Stimulation of vagus fibers supplying the pancreas causes an increase in insulin output, which produces a parasympathetic effect which again is met and counteracted by the sympathetico-adrenal mechanism, as previously mentioned.

ANALYSIS OF SYMPTOMS ACCOMPANYING TOXEMIA

Another very interesting study from the standpoint of symptoms is that of the toxic state. Toxemia is followed by a chain of many symptoms such as fatigue, chill, aching, fever, loss of strength, increased nervousness, insomnia, rapid heart action, dilatation of superficial blood vessels, sweating, dry mouth, lack of appetite, lessening of the secretions and the motor functions of the gastrointestinal canal, increased respiratory effort and either a leucopenia or a leucocytosis, according to whether the white cell regulating mechanism is depressed or stimulated.

If we think of these many symptoms as individual expressions of physiologic disturbance, we are confused, for individually they fail to give us any concrete picture of what is going on as a result of disease. If, however, we think of them as being symptoms called forth by a toxic state, we have made some progress; but are still far from having any definite intelligent grasp of what they mean. But if we think of them as being a part of the manner in which the body expresses its attempt to defend itself against injurious poisonous substances which are brought to it by an infection or by other means, a part of the defensive mechanism which has been called out, then we have a definite method of approach, but one which still requires a great deal more information before we can offer an explanation of their cause.

In attempting to account for these symptoms and make them understandable, the first thing that must impress a student of visceral neurology is that they are expressions of stimulation of the sympathetic nervous system and its inseparable adrenal gland.

This toxic state is very apt to be characterized in terms of temperature which is only one of its most prominent expressions. What should come to one's mind is that the toxic state calls out a general response on

the part of the sympathetico-adrenal system. The thyroid, too, must be considered as a part of this system. This is the particular system which has for one of its chief functions the preparation of the individual for physical defense and the sustaining of him during the period of struggle. So we now get a closer view of what our toxic symptoms mean. They are an expression of certain states which are brought about in the body in order to defend it against some poisonous substance; in the case of bacterial disease they are expressions which accompany the specific immunity response, and probably are necessary to its establishment.

This toxic reaction of the body differs somewhat but only in details of expression from the defense against outward foes. The difference seems to be due to the fact that the effect is released from within by a toxic chemical element. The general peripheral expression, however, is the same, consisting of those effects which are produced by stimulation of the sympathetico-adrenal system.

Cannon and his co-workers have shown that the bodily states which are called out by such major emotions as fear, anger, grief, preparation for fight or flight, and pain, produce their effects by stimulating certain nerve stations in the thalamus; so, too, because of the similarity of symptoms we must suspect this part of the brain to be the recipient and distributor of impulses in defense of the body in toxic states. The expression of anger and rage is a primitive function, likewise the function of defense was necessary and exercised before the cerebral cortex was developed; and while the cortex can inhibit to a certain degree some of the emotions which call out physical defense, yet such function is largely reflex in character and put into effect without volition. The defence against toxins seems to be wholly out from under cortical domination and in this way differs from physical defense.

With present knowledge, we are not in a position to state definitely that toxic effects may not be expressed in the cells of the body generally, aside from those produced through the sympathico-adrenal system; but from analyzing the effects shown, it seems quite clear that there is not only a lack of stimulation, but an actual inhibitory effect exerted by toxins on those structures which are activated by the partsympathetic system, as may be inferred from the effects shown on the heart, the salivary, gastric, intestinal, pancreatic and biliary secretions, on hunger and digestive peristalsis and on the bronchial musculature and glands. As a part of the toxic syndrome all of these systems are made conspicuous by the depression in activity which characterizes them. This indicates that toxic effects are expressed in sympathetic syndromes in the same manner as such major emotions as fear, anger, rage, pain and defense against outward foes, and such minor emotions as those caused by worry, discontent, unhappiness and pessimism.

The body's reaction to toxins, whether bacterial or nonbacterial, is always shown in the same way. Infections caused by pathogenic bacteria of all kinds are accompanied by a response which is qualitatively the same whether they be diphtheria bacilli, typhoid bacilli, streptococci, tubercle bacilli, or the causative germs of measles and smallpox.

It was suggseted by Vaughn several years ago that there are two factors in bacteria which produce reaction; the one specific and capable of stimulating the immunity mechanism of the host; the tother toxic and responsible for the toxic reaction. Further evidence sustaining such a conception has recently been adduced in the study of the tubercle bacillus in which it has been shown that a certain saccharid fraction which is attached to the protein molecule is responsible for highly toxic effects. Vaughn thought he had proved that the toxic element was not a carbohydrate, but more recent methods of study soom to indicate that the toxic element in tuberculoprotein at least is. Be this as it may, the usually recognized rise of temperature with malaise, aching, rapid pulse, and decreased activity in the glandular and muscular functions of the gastrointestinal canal consistently accompany immunity responses of the body to infections.

The toxic reaction may bring about conditions advantageous to specific defense even though not caused by specific immunizing substances. As a result of the reaction the patient's resistance to toxins is undoubtedly increased; from the rise in temperature the physico-chemical reactions of the body are quickened, and the immunity mechanism is permitted to act with great efficiency; and at the same time, the increase in the temperature of the tissues makes them unfavorable for the growth and multiplication of microorganisms.

It must be evident to the student of clinical disease that there is not a single symptom in the group of toxic origin that is pathognomonic of any particular disease. Measles, whooping cough, tonsillitis, typhoid fever, tuberculosis, pneumonia, malaria, and all other infections show similar toxic reactions, differing only in degree. The same temper-

ature and the same other symptoms may be produced by the injection of egg-white, Witte's peptone, or other protein substances. The curve of the temperature may be made to imitate the curve found in any of these infections by varying the dosage, in size and interval.

The important point regarding the toxic reaction is that it represents general nerve disturbance with a preponderantly peripheral effect expressive of stimulation of the nervous system. The same general symptoms are also found in such nervous states as neurasthenia and psychasthenia, also in many conditions significant of endocrine imbalance. Even a slight rise of temperature may be present in individuals who have an unstable nerve balance. Realizing that eighty-five per cent of the heat of the body is thrown off through the skin, and further realizing that a temperature curve only represents the balance between heat production and heat elimination, one may readily see how an individual might show a slight elevation of the temperature curve, if the vasomotors of his skin were slightly stimulated, even though there were no toxins set free in the body to produce the reaction.

It must be evident then that the members of the toxic group of symptoms are not of particular diagnostic worth, when considered apart from symptoms of the reflex and local groups. The temperature curve, however, varies according to the size and the frequency of the dose of toxins absorbed, which differs with different diseases to such an extent that the older clinical observers learned to associate definite types of temperature with definite diseases. Thus they recognized a certain type of temperature for typhoid fever, another for malaria, and still another for tuberculosis, et cetera.

A STUDY OF REFLEX SYMPTOMS

No group of symptoms is more fascinating for study than those of a reflex nature. Every organ is supplied by sensory or afferent nerves, and if it should become the seat of an inflammatory process, or if for any other cause, it should give origin to stimuli of undue strength, it has within it the possibility of originating reflexes. These reflex effects, if mild, may be dissipated without calling out any demonstrable changes in function of other organs or tissues; or, if severe, may cause serious and widespread disturbances in function.

For the purpose of producing reflex effects, every efferent nerve in the body may be brought into contact with impulses which are carried centralward over any afferent neuron, as must be evident in the universal spasms set up in the animal whose synapses have been affected by strychnin poisoning. A touch on any part of the body, a noise or a sight of something exciting may throw the animal into general convulsions.

Afferent nerves in internal viscera are not so necessary for defense as they are on the surface of the body which is exposed to constant danger of injury. The protected situation of the viscera does not call for quick response on their part to such stimuli as those of a cutting, burning, tearing or pinching nature to which the superficial structures of the body are exposed. Such stimuli as these, in the other hand, are commonly met by exposed portions of the body and require that special receptors for them be distributed everywhere over its surface in order that it may quickly respond with defensive reaction and avoid injury. Nevertheless, the viscera are supplied by afferent fibers, though relatively much fewer in number, which carry impulses back to the central nervous system and transfer them to other neurons through which action may be brought about. The effects so produced depend upon the severity of the stimulus, the frequency of its repetition, the time over which it extends, and the function of the neuron to which the stimulus is transferred. The effect may be contraction of a muscle; it may be a secretory effect; it may be a sensation; or, if the injury lasts a long time, it may be a trophic effect.

Many reflex effects are physiological and take place in conditions of normal health. A certain and important control of body function is due to the correlation of activity of separate and even widely separated structures, and this control is brought about normally by impulses which are picked up by the sensory organs in the tissues and carried centralward by afferent neurons and transmitted within the central nervous system to neurons which affect activity in the tissues of their peripheral distribution. In case of disease or injury of any of these organs the normal reflex pathways are made to carry the abnormal impulses generated by inflammation or other irritation, and, instead of there being a mild stimulation effecting a benign influence, there is an unusual stimulation with tendency to disturb and distort function; and, if continued sufficiently long, to work permanent injury to the organism.

These reflex effects become an important cause of symptoms in many diseases. Any disease which causes an inflammatory process in the tissues may cause reflexes. Hence reflex symptoms come to be an important part of all inflammatory diseases which are

localized in internal viscera. Reflex symptoms are also caused without inflammation being present whenever abnormal stimulation of afferent neurons takes place. In this way pain and cramps may result from abnormally strong peristaltic action in different portions of the alimentary tract.

Little progress can be made in the understanding of the clinical manifestation of such pathologic processes without a working knowledge of visceral neurology.

By knowing the innervation of the organs, and the segments in the central nervous system in which the majority of the impulses are most apt to form reflexes, and then by understanding the laws under which reflexes normally take place, as well as the laws which govern their variation, one will find that reflex symptoms of disease take on a new and definite meaning.

REFLEX EFFECTS FROM INFECTED TEETH

A toothache, if one understands reflex relationships, may become a very interesting study. During the toxic stage symptoms of sympathetic stimulation are prominent, being caused by both the toxins and the pain. But aside from that the reflex effects are very interesting. The teeth are supplied with sensory fibers by the Vth cranial nerve. The pain of toothache may be expressed in a wide area; first, in and about the tooth itself, and, second, over the superficial sensory fibers of the Vth nerve, particularly of the maxillary portion in case of an upper tooth and the mandibular portion if it is a lower tooth. Reflex lachrymation may be caused, particularly if an upper tooth is involved, by the impulse being carried centralward and then being transferred to the VIIth cranial nerve which supplies the lachrymal glands. Likewise, an increase of nasal secretion through the VIIth supplying the nasal mucous glands; and sometimes salivation through the VIIth and IXth nerves. Often, too, reflex nausea and vomiting are shown on the part of the gastrointestinal canal. Locally pus formation and exudative phenomena appear.

ANGINA PECTORIS

The symptoms of angina pectoris are also extremely interesting. The pain in the upper portion of the chest on the left side and in the left arm is the best recognized of all symptoms because it overshadows all else. It is regularly expressed in the Ist, IId and IIId dorsal sensory nerves, on the left side. These arise from the first, second and third thoracic segments of the cord. The heart receives its sympathetic nerve supply from the left half of the upper five or six thoracic segments; the ventricle particularly from the upper three. Since it is the natural thing for afferent impulses to be transferred to efferent nerves in the same segment which they enter, and on the same side of the body, the nerves arising from the upper thoracic segments and particularly those arising from the left half of these segments are the nerves which would be expected to express pain in case of heart lesions.

Sometimes, however, the pain is transferred upward into the neck, being expressed in the cervical sensory nerves and sometimes across to the right side of the body. All of this occurs, however, according to a definite law in physiology which governs the spread of reflexes upward or downward in the cord. This transference could probably occur either upward from the impulses which reach the cord in the upper thoracic segments or downward from those reaching the central nervous system in the medulla.

Spastic contraction of the muscles also occurs in angina, being produced through the motor nerves which correspond in origin to the sensory nerves which show pain.

There is also a reflex increase in respiratory effort, reflex effects in the gastrointestinal canal and reflex disturbance through the circulatory system, all of which follows regular paths over which physiologic impulses flow in conditions of health.

PULMONARY TUBERCULOSIS

The lungs offer an excellent opportunity to study reflex symptoms. They do not show pain like the hollow viscera such as the heart, stomach, intestine, gall-bladder, gall ducts and ureter, which have contraction as a part of their normal function; but by furnishing a large area which may be involved in the inflammatory processes, they furnish the ideal condition for originating many afferent impulses, particularly when infected with a widespread chronic inflammation such as is found in tuberculosis.

I have been able to suggest the paths for about forty reflexes from the lung, and have been able to prove most of them. The lungs being supplied by both the sympathetic system and the vagus of the parasympathetic system, and each of these systems having afferent neurons associated with them offer two distinct routes over which afferent impulses may travel; that over the sympathetic route goes to the upper five or six thoracic segments; and that over the vagus route to the medulla.

The afferent impulses which reach the central nervous system over the sympathetic route are transmitted up into the cervical segments of the cord, where they both mediate with motor and transfer their impulses

to sensory nerves going to the skin, subcutaneous tissue and muscles which arise from that region, particularly the third, fourth and fifth segments. These produce spasm of muscle, changes in sensation and degeneration of tissues. They likewise transfer impulse to sympathetic efferent neurons in the upper thoracic region which cause reflexes in the viscera.

The afferent impulses which course centralward with vagus are transferred in the medulla and midbrain to other cranial nerves, and produce widespread motor, secretory, trophic and sensory reflexes throughout the cranial region and in the viscera supplied by other branches of the vagus. Rasmussen and Larsell believe that the reflexes in the cervical portion of the cord also may be due to impulses transferred downward into the cord from the medulla. Through the vagus, such important structures as the larynx, heart and gastrointestinal tract, with the pancreas and liver, are affected.

The following four tables will show how widely spread the reflex effects from an organ may be and will serve as a paradign for studying other viscera.

TABLE I. PULMONARY REFLEXES IN SKELETAL STRUCTURES IN WHICH AFFERENT STIMULI COURSE CENTRALWARD OVER THE SYMPATHETICS AND EFFERENT OVER THE CERVICAL SPINAL NERVES PRINCIPALLY CIII, CIV AND CV.

I. Motor reflexes when the disease is active, as follows:

1. Spastic contraction of the muscles of the shoulder girdle (sternocleidomastoideus, scaleni, pectorales, subclavius, trapezius, levator anguli scapulae, rhomboidei) and the crus and central tendon of the diaphragm.
2. Limited motion of the half of the thorax, on which the diseased lung is found, through reflex spastic contraction of the sternocleidomastoideus, scaleni and subclavius above, and the crus and central tendon of the diaphragm below.

II. Trophis reflexes when the disease becomes chronic, or has healed, as follows:

1. Atrophy of the muscles of the shoulder girdle.
2. Atrophy of the skin between the second rib angle of the jaw anteriorly; and the spine of the scapula and the base of the skull posteriorly.
3. Atrophy of the subcutaneous tissue between the second rib and angle of the jaw anteriorly; and the spine of the scapula and the base of the skull posteriorly.

III. Sensory reflexes as follows:

1. Altered sensation, both superficial and deep, usually in the form of discomfort or aching rather than sharp pain; noted both when the disease is acute and chronic.
2. Vasomotor phenomena producing flushing of the ear through the 3rd cervical sensory nerve, when the disease is active.

TABLE II. PULMONARY REFLEXES IN SKELETAL STRUCTURES IN WHICH AFFERENT STIMULI COURSE CENTRALWARD OVER THE PARASYMPATHETICS (VAGUS) AND EFFERENT OVER THE VARIOUS CRANIAL NERVES.

I. Motor reflexes when the disease is active, as follows:

1. Spasm of the sternocleidomastoideus and trapezius, through the spinal accessory. (Possibly spasm of all muscles of the shoulder girdle, through cervical nerves, as suggested by Rasmussen).
2. Probably motor reflexes in the facial muscles through the facialis and trigeminus, since these atrophy when the process becomes chronic.

II. Trophic reflexes when the disease becomes chronic or has healed, as follows:

1. In the facial muscles through the facialis and trigeminus.
2. In the tongue, causing atrophy and deviation toward affected side, through the hypoglossus and lingual.

III. Sensory reflexes as follows:

1. Headache, face ache, etc., through the sensory branches of the trigeminus, both when the disease is acute and chronic.
2. Vasomotor phenomena producing flushing of the face, through the sensory fibers of the trigeminus when the disease is active.

TABLE III. VISCERO-VISCERAL PULMONARY REFLEXES IN WHICH BOTH AFFERENT AND EFFERENT STIMULI COURSE OVER THE SYMPATHETICS

I. Motor and secretory visceral reflexes when the disease is active, such as:

1. Dilatation of pupil.
2. Inhibition of motor and secretory activity in gastrointestinal canal.
3. Spasm of sphincters in gastrointestinal canal.
4. Increased heart action (tachycardia.)
5. Probably decreased motility and decreased secretory activity in ducts and glands of gallbladder, liver and pancreas, although not readily proved.

II. Probably trophic reflexes in structures subject to reflex action, when the disease is chronic or has healed.

TABLE IV. VISCERO-VISCERAL PULMONARY REFLEXES IN WHICH BOTH AFFERENT AND EFFERENT STIMULI COURSE OVER THE PARASYMPATHETICS.

I. Motor and secretory visceral reflexes when the disease is active, such as:

1. Increased secretion in the mucous glands of the nasal and naso-pharyngeal mucous membranes (catarrh) through the VIIth cranial nerve.
2. Disturbances in the motility of the vocal cords through the superior and inferior laryngeal nerves,
3. Probably motor reflexes in the lingual muscles through the hypoglossus since this atrophies when the disease becomes chronic.
4. Slowing of heart through the vagus.
5. Increased motility and increased secretory activity in gastro-intestinal canal through the gastric and intestinal fibers of the vagus, favoring appetite and digestion.
6. Probably increased motility and increased secretory activity in the ducts and glands of liver, gall-bladder and pancreas, favoring digestion, although not readily proved.

7. Cough, the afferent impulse being carried over the vagus to a center from which afferent impulses go out producing action in the abductor muscles of the larynx over the inferior laryngeal nerve, and in all expiratory muscles through the various spinal nerves; and simultaneously producing inhibition of action in the adductor muscles of the larynx and the muscles of inspiration.

II. **Trophic reflexes in structures subject to reflex action when the disease is chronic or has healed.**

1. In the tissues of the nasopharynx through the facialis.
2. In larynx through superior and inferior laryngeal nerves.
3. Probably in heart, gastrointestinal tract, liver and pancreas, though not readily proved, through vagus.

III. **Sensory phenomena.**

1. Laryngeal irritation through the superior laryngeal nerve.

These examples will serve to show how reflex effects may be unraveled by the clinician if only he will bear in mind the paths through which the visceral nerves have the power to correlate activity in different organs and structures.

The endocrine glands also come into the picture and take part in producing definite and well recognized syndromes at times.

Symptoms may originate, or may be changed, when established, by alterations in the chemical control of the body. A disturbance in the normal products of the various glands of internal secretions causes certain syndromes which are at times so definite that they can be recognized with ease and have come to be recognized as the clinical picture of increased or decreased function. Aside from this, each organ or function which depends on a normal secretion of some gland for its stability is thrown out of its usual mode of reaction under endocrine dysfunction and so effects symptoms that may be expressed in states of disease.

SUMMARY

(1) Pathologic physiology must be studied as intensely as pathologic anatomy, if we are to understand the symptoms of clinical disease.

(2) The true value of symptoms may be assessed only by classifying them etiologically.

(3) The relationship of symptoms to the vegetative nervous system is discussed.

(4) An etiologic classification of symptoms is offered for infectious diseases involving important organs, as follows:

(a) Group I. **Toxic symptoms** which are expressed generally throughout the body, but particularly throughout the sympathetico-adrenal system.

(b) Group II. **Reflex symptoms** which are produced by irritation of afferent nerves supplying a given organ which carry the impulse centralward and transmit it to some efferent neuron for action.

(c) Group III. **Local symptoms produced** by the inflammatory condition in the organ itself.

(5) The etiologic classification of the symptoms which present in a few widely different clinical conditions is discussed.

(a) Symptoms appearing with changes in blood sugar level.

(b) Symptoms accompanying toxemia.

(c) Symptoms accmopanying acute abscess of teeth.

(d) Reflex effects from angina pectoris.

(e) Reflex effects from pulmonary tuberculosis.

REFERENCES

Cannon, Walter B.: "Bodily Changes in Pain, Hunger, Fear and Rage." New York City, 2d edition, 1929, D. Appleton and Co.

Vaughn, V. C.: "Protein Split Products in Relation to Immunity and Disease." Philadelphia, 1913, Lea and Febiger.

Pottenger, F. M.: "Symptoms of Visceral Disease," St. Louis, 4th Edition 1930, C. V. Mosby Co.

Pottenger, F. M.: "Principles Which Govern Reflex Action in Disease." Transactions of the Amer. Therapeutic Society, Vols. XXVIII and XXIX, 1927-1928.

Mackenzie, Sir James: "Symptoms and Their Interpretation. "London, 1909. Shaw and Son.

Mackenzie, Sir James: "The Future of Medicine." London, 1919, Oxford University Press.

Pavlov, I. P.: "Conditioned Reflexes." Translated by B. V. Anreb. London, 1927, Oxford University Press.

Bauer, Julius: "Die Konstitutionelle disposition zu Inneren Krankheiten," Berlin, 1921, Verlag von Julius Springer.

Pende, N.: "Constitutional Inadequacies." Translated by Sante Naccarati, Philadelphia, 1928, Lea & Febiger.

Falta, Wilhelm, "The Ductless Glandular Diseases". Translated by Milton K. Meyers, Philadelphia, 1916, P. Blakiston's Son & Co.

Barker, Lewellys F., Hoskins, R. G., and Mosenthal, Herman O.: "Endocrinology and Metabolism," New York, 1922, D. Appleton and Co.

Cramer, W.: "Fever, Heat Regulation, Climate, and the Thyroid-Adrenal Apparatus." London, 1928, Longmans, Green and Co., Ltd.

Sherrington, C. S.: "The Integrative Action of the Nervous System, New York, 1906, Charles Scribner & Sons.

Pottenger, F. M.: "The Syndrome of Toxemia—an Expression of General Nervous Discharge through the Sympathetic System. Jour. of the Amer. Med. Assn. Jan. 8, 1916, Vol. LXVI, pp. 84-85.

Pottenger, F. M.: "Some Practical Points in the Diagnosis of Active Tuberculosis." Northwest Medicine, January 1914.

DIAGNOSIS OF TUBERCULOSIS IN CHILDREN

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(Address of the fraternal delegate from Texas, to the Arizona State Medical Association, at their Thirty-ninth annual meeting, in Phoenix, April 24 to 26, 1930).

Diagnosis of tuberculosis in children is, to my mind, extremely important for three reasons: (1) to enable the condition to be controlled in order that the child may not be destroyed or maimed, (2) to protect other children from over-exposure to the disease, and (3) as a means of approach to check adults and other members of the family, to find the sources of infection. And diagnosis of the childhood condition is by no means easy, since we find so many cases in which there are no manifest symptoms when the disease is far advanced, and, on the other hand, other cases which have several noticeable symptoms but are entirely devoid of apparent physical signs. Our problem has developed almost far enough to cause us to consider every child infected unless we can prove otherwise, especially if there is a case of adult tuberculosis in the family.

H. W. Hetherington (American Review of Tuberculosis, 1927) reports tuberculin tests on 1999 school children and finds that about seventy-two percent of them reacted positively. Their age range was from five to sixteen years and he found that reactors increased in numbers with the age, 35.5 per cent at five years of age to 93.2 percent at fifteen years of age. To my mind, this means nothing more than that children in the cities and in school are constantly exposed, and the longer they are exposed the more likely they are to contract the disease.

It is rather hard for us to imagine that thirty-five percent of the five-year-old children whom we see are diseased; or seventy-five of the ten-year-olds; or ninety-three percent of the fifteen-year-olds; but such is the case. True, only a small percentage of these later develop the disease further and are destroyed by it; but for the reasons named we should be more careful in our examinations and try to diagnose the condition early enough to enable us to come nearer the goal of obliterating the disease entirely.

In recent years so much attention has been given to food and milk production that only a small percentage of infection can be laid to these as sources. By far the largest percentage of infections come through the respiratory tract from an open case in the family. For this reason I say that all children in a family where there is an open case of tuberculosis should be treated as if they were infected, whether there are symptoms or signs, or neither.

The usual type of tuberculosis found in children is termed the childhood type and differs materially from the adult type. It starts, in most cases, with a formation within the parenchyma near the periphery in any part of the lung, of one or more tuberculous nodules with caseation in the center. In the favorable cases, usually in older children, these nodules become encapsulated and remain small, but in others there is a peripheral spread and involvement of more of the lung tissue. In the favorable cases there is a later deposit of calcium in the nodule coincident with the encapsulation. While this is going on, there is a drainage to the tracheobronchial nodes and these become infected in varying degrees. There may be single or massive infection of these nodes, which may break down entirely but, in favorable cases, become calcified.

In the adult type, which may be found in children but always with a grave prognosis, the infection is secondary, exogenous or endogenous, and almost always starting at the apices. The tracheobronchial glands are rarely infected in this type, and caseation is, in most cases, followed by cavities instead of calcification.

The diagnosis of tuberculosis in children depends on a number of factors, of which the most important are: (1) history; (2) symptoms; (3) physical signs; (4) laboratory tests, such as x-ray, tuberculin, complement fixation, etc.; and the most important of these is history. The physician should carefully question the child or his parent to find out whether or not there is a case of adult tuberculosis in the household. Henry D. Chadwick, in a report of examination of 25,000 Massachusetts school children, found that four times as many cases of childhood type and twice as many cases of adult type were found in children said to have been exposed to a case of pulmonary tuberculosis, as among children not exposed. In so many cases where the history of an open case in the family is denied, a careful study and questioning will reveal a case who has had chronic bronchitis for years. Usually these cases turn out to be fibroid phthisis and constant exposure is taking place. Not always do we find that a few exposures to an open case will cause the disease in childhood, but constant, close contact will, without doubt, cause the childhood manifest type, and in many cases the adult type. Infection is probable in the schools through teachers, where the city health board is lax in its examina-

tions, and also where the supervision of childhood health is not thorough and there are open cases without severe, manifest symptoms in one or more children.

By no means should the production of food and dairy products be overlooked as a source of infection, especially in the smaller towns where the dairies are not under close supervision of the city or county health boards. Due to the prevalence of tuberculosis in cattle, the children should be safeguarded from the bovine type by being fed only pasteurized milk. Special care should be taken to investigate the cook and nurse of the child to see that the food is not contaminated at this source.

Symptoms may be many and varied, or entirely lacking. In some children we may find the childhood type without any symptoms that can be ascribed to the disease, even though the disease is progressive at the time. Tuberculous infection should be looked for in the child who is slightly hoarse, has frequent colds and sore throat and cough, especially if the cough is productive. In children, frequent upper respiratory infection is of much more significance than in adults. The weight question does not play such an important role, since childhood tuberculosis is almost as prevalent in overweight as in the underweight child; and we readily see by this that, if we examine only the underweight children, we shall miss a great many cases.

The child who tires easily and loses his natural inclination to play with other children, should be watched closely. I am always suspicious of the child prodigy who is extremely bright and leads his class in school, often being a grade or two ahead of the other children his own age. In many cases we learn that he spends very little of his time out of doors and much of it with his books because he is too tired to play. I believe, also, that a small amount of absorption from tuberculosis is a mild, mental stimulant, enabling the child more quickly to grasp his problems and learn his lessons.

An occasional rise in temperature to 99.5° or 100° cannot be taken as an evidence of disease. The normal temperature range in children is much more variable than in adults, and any active exercise or close confinement in a warm room will bring about an increase in temperature of one to one and one-half degrees. However a persistent daily rise to 99.5° or100° is sufficient cause to make necessary a careful search for disease. Often a sudden rise in temperature which lasts for a few days and then subsides is caused by a pleuritic effusion. Physical examination and roentgenograms can confirm the diagnosis, and unless some other cause is evident, pleural tuberculosis should be considered as the source.

Only a few of the phases of childhood tuberculosis can be detected by physical examination. Enlarged and matted cervical glands should be considered as evidence, but glands which are simply palpable can be ignored. Tracheobronchial gland enlargement can rarely be detected by percussion, which is also the case with the small pulmonary nodule. Auscultation and percussion are of service only in detecting the diffuse infiltrations in the lung.

The tuberculin skin test is by far our most reliable source of information in detecting tuberculous infection. A positive reaction always means that the infection is present even though it does not indicate whether the disease is progressive or latent. There are two methods in general use; the Pirquet method. which consists of scarifying the skin and allowing the tuberculin to absorb; and the Mantoux, or intracutaneous method. The latter is more accurate and a greater number of reactors will be found if it is used. A negative skin reaction with as high as 1.0 mg. of tuberculin, unless in the presence of one of the severe exanthemata, means absence of infection.

The complement fixation test is of doubtful value, but Dr. George Turner, of El Paso, is doing an extensive amount of work on it at present in connection with his experiments in deproteinizing blood serum, and it is to be hoped that his work will be of value to us in diagnosing early tuberculosis.

Every child who reacts positively to tuberculin should have a chest roentgenogram. In only a comparatively few cases will childhood tuberculosis be evident on a film, but no diagnosis can be complete without a careful x-ray study. Diffuse infiltration will show in any part of the lung and have the appearance of tuberculous broncho-pneumonia. As these areas are re-absorbed, there remain only small calcified nodules or strands which may in turn disapper at a later date. Tracheobronchial node infection is more or less evident, depending upon the amount of calcification which has taken place. The enlarged glands in which there is no calcification cannot be differentiated from the surrounding tissues, and a diagnosis can be made from them only when the increase in size is enough to cause them to protrude as rounded masses beyond the hilus. In some cases, if the picture is made with the right side of the patient turned slightly away from the film, these glands

will be brought out more clearly. Blood vessels caught in cross-section and bronchi are sometimes mistaken for enlarged nodes.

The parenchymal lesions have to be differentiated from such pulmonary conditions as lung abscess, bronchopneumonia, and bronchiectasis. In abscess there is usually the history of a recent upper respiratory operation, or the possibility of a foreign body, to be considered. Broncho-pneumonia has a history of an acute illness, and the lung density following it clears rapidly. Bronchiectasis has, in nearly every case, broncho-pneumonia as a forerunner, and we find the symptoms of the latter all disappearing except the persistent cough and sputum. Tracheobronchial infection may be confused with Hodgkin's neoplasms, or enlarged thymus, but usually a careful study or angle picture will clear these up.

In conclusion, I wish to present the plan in use in the schools in Massachusetts to find the cases of tuberculosis in children which should be under observation and treatment. It is, I think, the most thorough and, at the same time, most economic plan I have come in contact with for discovering tuberculosis in groups of supervised children. Every school child is subjected to tuberculin test and at the end of five days the readings are completed. All non-reactors are dismissed and the reactors then have x-rays made. All cases who do not show any abnormal shadows in the film are dismissed and the others are brought in for a careful physical examination. This plan is effective in that it locates all cases of tuberculosis so that they can be observed and given a bit more care, and also differentiates the cases needing only observation from the ones needing closer observation and treatment. Another good point to recommend it is the fact that the examining personnel can be small.

ESOPHAGEAL DIVERTICULA

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(Read, by invitation, before the thirty-ninth annual meeting of the Arizona State Medical Association, held at Phoenix, April 24 to 25, 1930.)

Esophageal diverticula, while probably not occurring any more frequently than formerly, due to a better understanding of this condition by the profession, are being diagnosed more frequently.

In 1840 Rokitansky[1] classified esophageal diverticula into two types, the traction and the pulsion type. This classification is still used. In 1881, Zenker and Ziemssen[2] described the condition known as Zenker's pharyngo-esophageal diverticulum. Oekenomides[3], in 1882, added the traction-pulsion variety.

Traction diverticula usually occur in the thoracic portion of the esophagus. They are caused by cicatricial contraction of a chronic inflammatory process drawing the wall of the esophagus outward and include the whole wall of the esophagus. The apex of the pouch is usually higher than its point of entrance into the esophagus and, therefore, it rarely attains any appreciable size or produces any symptoms. Occasionally food particles may lodge in the pouch. The sac then enlarges, due to pressure from within, and in this manner a traction-pulsion diverticulum is formed. Judd[4] estimates that this occurs in about seven percent of traction diverticula. He reports two personal cases. Case No. 1 of this series also belongs to the traction-pulsion type.

Pulsion diverticulum usually occurs in elderly people, mostly men, and always occurs in the cervical region. The point of origin is fairly constant, directly back of the cricoid cartilage on the posterior wall of the esophagus, at its juncture with the pharynx. At this point, where the oblique muscles of the pharynx and the transverse circular muscles of the esophagus meet, there is a small triangular area not covered by muscle. This area has been described by many writers as the Lannier Hackerman area.

Bevan[5] believes that a predisposition to the development of diverticulum may occur as the result of more than normal weakness at this point, possibly due to a congenital absence of muscle fibers over a large area. Judd, in reviewing his cases, could find no clue to the etiology other than weakness in the wall of the upper end of the esophagus. There had been no history of trauma or any evidence of anything unusual in mastication or deglutition. Jackson[6] believes that the chief factor in the etiology of esophageal diverticulum is the pinch-cock action of the coricopharyngeus muscle. Normally the pinch-cock is always closed except when it momentarily opens upon the approach of food or water. When it fails to open, a tremendous pressure is exerted on the pharyngeal walls, which causes pouching at the weak point. He recommends gently stretching the cricopharyngeal constriction, even when not organically strictured, to lessen the tendency to recurrence of the diverticulum, and to prevent post-operative leakage.

Morley[7] states that there is no evidence of a congenital origin and states that we can only assume that, for some unknown reason, the circular fibers at the upper end of the esophagus fail to relax in the process of de-

glutition. Durr[8] reports that half of his cases of pulsion diverticulum were associated with goiter. Von Hofmeister[9] also reports that in a series of nine cases, five had retrosternal goiters. Bull[10] reports that diverticulum of the esophagus has been observed in association with idiopathic dilatations of the esophagus.

Whatever may be the etiologic factor, once the esophageal muscles have been penetrated the sac gradually enlarges. At first the pouch is in mid-line growing down behind the esophagus, gradually increasing, usually into the left side of the neck. In this series of ten cases, the pouch presented in the right side of the neck four times. As the sac gets larger it frequently grows into the mediastinum, becoming intrathoracic. The walls of the sacs are very thin and are composed of a layer of mucous membrane and fibrous tissue. Judd has called attention to the fact that, while occasionally there may be a band of muscle fibers extending across the sac, muscle tissue is not a part of the sac wall.

The inception is probably rarely recognized, as the sac has to attain some size before it will give trouble in swallowing. Dysphagia and regurgitation of food are usually the first symptoms. Later, gurgling noises in the throat and cough are complained of. As the sac increases in size, dysphagia is more marked, resulting in loss of weight.

The diagnosis is not difficult. No complicated tests are necessary. Given a patient with a history of dysphagia and regurgitation of food, the first thing to order is a barium meal examination. The x-ray will always reveal the sac if the condition is due to a pulsion diverticulum. The x-ray will also determine in which side of the neck the sac presents. In the patients with a large sac, one can usually see one side of the neck enlarging as the patient drinks water and, by making pressure over this area, the fluid can be made to regurgitate back into the mouth. The diagnosis is never complete without an esophagoscopic examination, for this will demonstrate or rule out other pathological conditions of the esophagus, such as organic strictures or carcinoma below the diverticulum.

The treatment of pulsion diverticulum is surgical, even in cases with small sacs which cause but little trouble. For at this time the patient's general condition is excellent. As time goes on the tendency is for the sac to increase in size, causing marked deformity of the esophagus. The result is an increase of dysphagia and regurgitation of food, resulting in a marked loss of weight and a patient who is a poor surgical risk.

Patients formerly came for treatment in a starved condition, and many required a preliminary gastrostomy for forced feeding. Fortunately, at present this is rarely necessary and in many cases the condition is recognized so early and the condition of the patient so good that no preliminary measures are necessary.

In patients who are in fair physical condition it is my custom to hospitalize the cases for a few days and force fluids both subcutaneously and per rectum. On the day preceding the operation, the patient is given intravenously 1000 c.c. of ten per cent solution of glucose. A duodenal catheter can frequently be passed through the nose, down the esophagus, into the stomach and feeding given through the catheter, which may be left in place from seven to fifteen days. When the sac is very large, it is almost impossible to pass a catheter into the esophagus. In this type of case Vinson[11] suggests having the patient swallow a thread and, using this as a guide, to pass the catheter as described by H. S. Plummer[12]. The catheter can be removed daily but not the thread.

All patients should be instructed to keep the sac empty for several days before the operation. This can easily be done by drinking several glasses of water soon after eating, thereby washing out the sac.

Many different methods have been used to treat this condition. Bell, in 1830, recommended an external fistula. Konig recommended elevating the sac and fixing its fundus to the hyoid bone. Wildenberg, in seven cases, changed the direction of the sacs, later removing three of them. Liebel fixed the sac to the skin incision, leaving it unopened. Von Beck recommended ligating the neck of the sac and leaving it to slough out of the packed wound. He reported two deaths from pneumonia in seven cases. Goldman reported, by Von Beck's method, sixty-five percent of fistulas with infection and slow healing. Niehaus probably made the first extirpation. Girard[13] in 1896, invaginated the sac and sutured the esophageal walls. Havan[14], elaborating this procedure, invaginated by suture and recommends in large sacs to remove a portion befroe invaginating. Murphy recommended delivering the sac unopened through the incision, twisting it, later amputating and closing the fistula. By this method many sacs became gangrenous, due to the interference to the circulation. C. H. Mayo[15] suggested sewing the sack, untwisted, to the sterno-mastoid, leaving it in this position for ten days, later amputating and closing the

opening in the esophagus. This is the method used today.

At present the operation is very well standardized. The only controversy existing is whether the operation should be completed in one stage, that is, freeing and excising the sac in one operation, or doing a two-stage operation, freeing the sac and suturing it unopened in the wound, with excision about ten days later.

Jackson[17], Shallow and Gaub[18], believe that the one-stage operation, with the aid of the esophagoscope and the use of the Rehfus tube post-operatively, is safer and gives much better results.

In cases with small sacs, surgeons have a choice. In this type of case the one-stage operation is excellent, but in large sacs extending into the mediastinum, with patients in poor physical condition, one has no choice —the two-stage operation is the only one to use.

The early operations for removal of diverticula were attended by high mortality. This mortality was due to infection in the neck, which usually extended down into the mediastinum. In the two-stage operation there is usually an interval of about ten days between the first and second operation. During this time the mediastinal space becomes closed by granulation tissue. This minimizes the danger of mediastinitis. Another advantage is that, by changing the position of the sac, it is no longer in direct line of the descent of food and patients are able to swallow without difficulty. Patients can usually be out of bed.

The operation should be performed, if possible, under local anesthesia, usually one per cent novocain. The sacs in many cases, especially if small, are very difficult to locate. A conscious patient is told to swallow and this causes a bulging of the sac which greatly aids in its location.

Whether one performs the one or two-stage operation, the technic of freeing the sac is the same, viz: The skin incision, including the platysma, is made along the anterior border of the sterno-mastoid from just above the level of the cricoid cartilage down to the clavicle. The sternomastoid is retracted outward and the deep cervical fascia is divided parallel with the skin incision. The large vessels of the neck are retracted outward and the sterno-hyoid muscle is retracted inward. The middle thyroid vein, if present, is ligated before any attempt is made to expose the thyroid gland.

The omohyoid muscle should be divided if it interferes with good exposure. The thyroid gland is rotated inward; this exposes the upper part of the trachea at its junction with the thyroid cartilage. Behind this may be seen the esophagus as a flat fold pressed against the vertebra. If the sac is small it will be behind the esophagus, slightly to the left of midline, just below the level of the cricoid cartilage. If large, it is very easily recognized. If local anesthesia is used, the patient is asked to swallow, the sac can be seen bulging out of the lower portion of the neck. Jackson has suggested a very ingenious method of localizing the sac. During the operation he inserts a small endoscope into the sac, the illumination from within guiding the surgeon.

The sac, when located, can usually be freed from the surrounding structures with blunt dissection. Some few firm adhesions may have to be cut. Great care should be taken not to perforate the sac. As soon as the sac is delivered from its position in the chest, the cavity should be packed with a warm sponge to protect the mediastinum from accidental leakage. One point of importance in the dissection is the danger of injuring the sac at its juncture with the esophagus. If the sac has a narrow neck, it is easy to differentiate between the esophagus and the diverticulum. However, if it has a wide opening into the esophagus, as is common, there is great danger of opening the sac or injuring the esophagus, depending on whether a one or two-stage operation is to be performed. The one-stage operation as performed by Gaub, Jackson and Shallow, is as follows: The neck of the sac, as in inguinal hernia, is transfixed with a fine needle and No. 1 iodized catgut. The sac is then cut away. Three interrupted sutures are now placed, each suture picking up the muscular coat of the esophagus. These sutures cause inversion of the stump. The esophagoscope being in situ during the operation, there is no danger in narrowing the lumen of the esophagus too much. An incision in the neck is closed in layers. A small piece of cello silk is placed beneath the deep fascia. The Rehfus tube is passed through the nose into the stomach and retained for from ten to eighteen days. This affords rest to the esophagus.

The two-stage operation as performed by C. H. Mayo, Judd and Lahey[19], is as follows: After freeing the sac from the surrounding structures and down to the esophagus, the sac is sewed to the sterno-mastoid or the platysma with a few catgut sutures. In doing this, great care should be taken not to pull out the sac too far, so that the esophagus will not be pulled out of normal position. Also, in sewing the sac to the muscle, some

sacs are so thin there is danger of penetrating the sac and causing a leak with secondary infection of the tissues of the neck. The wound in the neck is sewed around the sac and the entire sac allowed to prolapse from the wound.

The sac is left in this position from eight to twelve days, depending upon its size, at which time the second operation is performed. The tissues around the neck of the sac are injected with one per cent novocain solution. The neck of the sac is again freed, either a purse string or a transfixing suture is used to tie it off, and the sac excised. A few interrupted sutures are used to reinforce the esophagus. A small fistula usually forms although it heals completely in a short time.

CONCLUSIONS.

1. The most important etiological factor in the production of pulsion diverticulum is the incoordination of the cricopharyngeal muscle, plus weakness of posterior muscles.
2. All patients, especially elderly people, complaining of dysphagia or regurgitation of food should have a barium meal examination.
3. Patients with diverticulum of the esophagus should be craefully examined with the esophagoscope.
4. Gastrostomy is rarely indicated, since forced feeding can be carried out by means of the duodenal catheter.
5. The two-stage operation is the safer procedure since it eliminates the danger of mediastinitis.
6. Local anesthesia is preferred.
7. In this series of eleven cases, nine males and two females, the average age was sixty-six.
8. There were no fatalities nor recurrences.

CASE NO. 1.

Female, H. W., age seventy-five, seen in consultation with Dr. Harold Smith. Chief complaint: loss of appetite and increasing constipation. Bronchial cough for four weeks. Always enjoyed good health until last eight years during which she has gradually grown weaker. Physical signs of diffuse bronchitis.

X-ray examination: shows a fairly good-sized diverticulum. Its lower border is about 10 cm. below the upper level of the arch of the aorta or 16 cm. below the upper border of sternum. The opening into esophagus about 3 cm. in width from above down, pouch itself extending one cm. above and below and 3 cm. deep. Posteroanterior stereoradiographs of the chest, made erect, show small chest, diaphraghms which do not move freely. There is a calcification within the arch of the aorta. There is some thickening of the lung roots with marked prominence of the descending branches, and some thickening in each hilus, particularly notable in the oblique views from the fluorscope.

No esophagoscopic examinations made.

Diagnosis: Traction pulsion diverticulum.

No treatment, as patient has no symptoms referable to the diverticulum. Dye series of gallbladder shows stones.

CASE NO. 2.

Male, F. S., age fifty-eight. Chief complaint: choking sensation and difficulty in swallowing, started seven years ago. At first trouble was intermittent, lately continuous. Has almost a constant desire to swallow and complains of gurgling noises in the throat. Has lost forty-eight pounds, mostly during the last two years. Otherwise examination negative.

X-ray examination: shows a large diverticulum of the esophagus, presenting in the left side of neck. Esophagoscopic examination negative for other pathology.

Preliminary treatment: Unable to pass a duodenal catheter into stomach due to large sac but succeeded in having patient swallow thread. Catheter passed over thread and forced fed for ten days.

Operation: local anesthetic one per cent novocain. Two-stage operation performed. A sinus persisted for two weeks. At present, four years since operation, perfectly well. Has gained fifty pounds.

CASE NO. 3

Male, D. O., age sixty-eight. Chief complaint: Difficulty in swallowing and regurgitation of food. Started six years ago, much worse last six months. Complains of gurgling noises in throat after eating. Lost only five pounds.

X-ray examination: shows a small diverticulum of the esophagus, sac practically in mid-line. Esophagoscopic examination was negative for other pathology.

No preliminary treatment, general condition excellent.

Operation: Anesthetic, nitrous oxide with oxygen. Two-stage operation performed. Sinus completely healed in four days. Three and one-half years since operation, no symptoms.

CASE NO. 4

Male, H. B., age sixty-two. Chief complaint: Difficulty in swallowing all kinds of food. Regurgitation and vomiting. Trouble started fifteen years ago. Has gurgling noises in throat after eating, also a troublesome cough. Has lost twenty pounds. Right side of neck bulged when patient drank water, sac held twelve ounces.

X-ray examination shows a large diverticular sac presenting in the right side of neck. Esophagoscopic examination by Dr. Jesberg was negative for other pathology of the esophagus. Five years previous an operation had been performed for the removal of the diverticulum, which was not successful. He had a discharging sinus for seven months. As soon as the sinus healed all of his symptoms returned.

Preliminary treatment: Fluids were forced for two days and 1000 c.c. of a ten per cent glucose solution was given intravenously day preceding operation.

Operation: Anesthetic, nitrous oxide and oxygen. Two-stage operation performed. Adhesions of sac very dense, due to previous operation. Sinus lasted three weeks. Three years since operation, no recurrence of any symptoms.

CASE NO. 5

Male, T. T., age seventy-four. Referred by Dr. John Barrow. Chief complaint: difficulty in swallowing. Had sour regurgitation but no vomiting. If he stooped over after eating, food ran out of nose. Has gurgling noises in throat after eating. Trouble started one year ago. Has had a severe cough for the last five years. Prostatectomy two years ago, history otherwise negative.

X-ray examination: shows a small diverticulum of

the esophagus presenting in the right side of the neck. Esophagoscopic examination by Dr. Jesberg revealed no other pathology. No preliminary treatment.

Operation: Local anesthetic one-half of one percent novocain. Two-stage operation. Good recovery. Sinus lasted five weeks. Very troublesome cough which might account for sinus remaining open so long. Eleven months since operation, no return of symptoms.

CASE NO. 6

Male, J. W., age seventy-two. Referred by Dr. J. Mark Lacey. Chief complaint: Ten years ago had difficulty in swallowing, feels as if food stopped in middle of chest and relieved only after a severe coughing spell. Last two years had regurgitation of food and vomiting, also gugrling noises in throat after eating. Blood pressure 225/140. Prostatectomy ten years ago, otherwise history negative.

X-ray examination: shows medium sized sac presenting on the left side of the esophagus. An esophagoscopic examination by Dr. Jesberg was negative. No preliminary treatment used.

Operation: Local anesthetic one-half of one percent novocain. Sac extremely thin, which ruptured during the dissection, frothy mucus escaped. Wound thoroughly irrigated with Ringer's solution. Sac then removed in one-stage Gaub Jackson technic. Small rubber tissue drain inserted into lower angle of wound. Patient unable to tolerate duodenal tube for more than three days, after which swallowed without difficulty. No sinus developed in spite of a very severe infection of the neck. This infection cleared up in two weeks. One year after operation, completely well.

CASE NO. 7

Male, E.M.L., age sixty-eight. Chief complaint: Regurgitation of food and vomiting. Started two years ago. Condition had gradually become worse. General health excellent. Previous history shows nothing bearing on present trouble.

X-ray examination: shows a medium-sized sac presenting in left side of neck. No other pathology of esophagus found.

Operation: Local anesthetic, one percent novocain. Two-stage operation. No sinus developed. Patient at present completely relieved of all symptoms (six months after operation).

CASE NO. 8

Female, Mrs. C.F.S., age forty-seven. Chief complaint: Dysphagia, spitting up of food, noise in throat, coughing and constipation. These symptoms started seven years ago. First noticed gurgling noises in throat while eating. Symptoms have increased until the present. Now she is very weak, gets very little food into stomach. Has lost fifty-one pounds. Blood sugar 62 mg. per 100 c.c. of blood. Examination otherwise negative.

X-ray examination: Shows a large diverticulum of the esophagus presenting in the right side of the neck. Esophagoscopic examination negative for other pathology.

Preliminary treatment: Patient hospitalized for four days. Was given salt solution subcutaneously and five per cent glucose solution per rectum and the day before the operation 1000 c.c. of ten percent glucose solution was given intravenously.

Operation: Local anesthetic, one per cent novocain. Sac was very adherent and extremely thin so that we were unable to do a two-stage operation as, in the attempt to sew the sac to the muscle, the stitches tore the sac. The one-stage operation was then done. The duodenal tube was immediately inserted and patient fed in this way for about fifteen days. At the end of seven days patient was able to swallow a small amount of liquids without difficulty so that at present no difficulty at all. Moderate drainage from wound for ten days which gradually healed up. Small sinus for four days.

CASE NO. 9

Male, J.H., age sixty-two. Chief complaint: Difficulty in swallowing, regurgitation of food, choking sensation and cough. Symptoms first noticed thirteen years ago; while eating some berries, noticed he could not completely swallow them. Then noticed difficulty in swallowing and regurgitation; could not swallow solid food such as bread, cheese, apples, etc. They all caused itching and choking sensation. Loss of weight was not marked, twenty-two- pounds in ten years. Previous history negative. Urine negative. Blood: Hemoglobin: 81 per cent, R.B.C. 4,752,000. Color index. .8 per cent. Leukocytes 9,000; polys, 66 percent; lymphocytes, 30 percent.

X-ray examination: Showed a large diverticulum lying posterior to the esophagus. It apparently comes off about on a level with the sixth cervical. It is oval in shape and fills before any of the meal goes into the distal portion of the esophagus. In size is approximately four centimeters broad and nearly eight long, and is in the center line. No preliminary treatment.

Operation: First stage under one percent novocain, local anesthetic. Sac was freed and sewed to the sternocleidomastoid muscle. Practically all of the sac was in the mediastinal cavity. Quite adherent to the upper part of the under surface of the thyroid. There was a small plexus of veins covering the sac, which had to be ligated. Patient did very well, was out of bed on the second day, able to take soft foods without any difficulty and all the liquids we wanted him to take. Second stage of operation was done eleven days later. The sac was freed down to the esophagus and transfixed with a suture, and three sutures of chromic catgut used to invert the stump of the sac into the esophagus. Post operative course was entirely satisfactory. Patient had no leakage whatever.

Patient was re-checked April 3, 1930, is in perfect health, working every day, absolutely no recurrence of any symptoms.

CASE NO. 10

Female, Mrs. F. B., age sixty-five. Chief complaint: Dysphagia, regurgitation, great loss of weight, swelling in left side of the neck, constipation, fatigue. Symptoms first noticed twenty-five years ago. Patient vomited quite suddenly some of the food, only part of the food would be regurgitated, and only after eating several times in a row would she get enough food to satisfy the appetite. Would eat as many as eight and ten meals a day. Type of food made no difference, liquids came back just as quickly as solid foods. All signs and symptoms have become accentuated in the last year. Recently, during the past twelve weeks, patient has hovered near death due to starvation and general debility. For several weeks was in the Hollywood Hospital, getting fluids by skin, vein and rectum. Has greatly improved.

X-ray examination: Patient was given a barium meal but only a small portion was seen to trickle down into the lower esophagus. The greater portion of the barium passed into a large, pear shaped sac lying behind the upper part of the sternum and extending over to the right side of the chest.

Preliminary treatment: Patient was kept in the hospital about five days before operation. During which time she received, daily, glucose intravenously and about one quart of salt solution rectally or subcutaneously. This improved the patient very materially.

Patient is an elderly woman lying in bed very emaciated. Takes very little interest in her surroundings. All the great vessels of the neck and face are somewhat distended when not swallowing food or liquid and when she drinks about sixteen to twenty ounces, the veins over the left eye, left cheek, left cervical region and all down the left arm distend very greatly, and are very varicose. Varicosities also show up when the patient does not swallow. The patient, on the left side of the face, when the sac is full, looks like a patient who is choking to death, especially the left eye seems to bulge out.

Operation: First stage, local anesthetic, one percent novocain. Sac was freed, sewed to the sternomastoid muscle and skin, and left outside of the neck. Practically all of the sac was in the mediastinum. The sac was very large, the walls were very thick, and there was a layer of muscle over the anterior wall of the sac. Under the thyroid, near the neck of the sac, was a plexus of veins. The sac was densely adherent to the vertebral fascia.

Following this operation the patient was able to swallow liquids and soft foods. The sac was very large, and three days later it became distended with air, causing the patient considerable pain. It had to be opened with a pin-point electric cautery, allowing the air to escape and the sac flattened out.

Things went along fairly well for about seven days, and then the patient developed a very severe ulcerated stomatitis, which prevented her from taking any liquids whatever.

Due to the patient's poor physical condition it became necessary to do either a gastrostomy, or some other type of forced feeding. I decided to cut off the sac, leaving about an inch protruding from the skin edges, and through this a catheter was inserted into the esophagus, and the patient was fed in this way without any difficulty. This was kept up for about eight weeks. The stomatitis cleared up after about three weeks. Patient then started taking a little food and liquids by mouth, but we kept up the forced feedings.

She gained in weight and her general condition improved. In the mediastinal area from where the sac was removed,—due no doubt to the number of attacks of inflammation, thickness of the sac wall and adhesions, and the length of time the sac was present,—a cavity remained, which would fill up with mucus and pus, and cause the patient a great amount of trouble and a cough. For this condition we inserted a catheter, and connected it with a continuous suction apparatus, and thus kept the cavity empty; we also irrigated it with Dakin's solution. This eventually cleared up after about eight weeks, and the patient was able to take food. We were then able to do the second stage of the operation, using Lahey's method of dissecting the mucous membrane down to the esophagus, leaving the outside fibrous layer. This sinus was packed daily and kept clean. Our reason for doing this was due to the fact that the walls of the sac were at least one-quarter inch thick, and it would have been impossible to invert the neck of the sac into the esophagus.

The patient left the hospital on the tenth week in fair condition considering the fact that she was brought into the hospital in a morbid and starving condition.

Three months after the operation, the patient developed a broncho-pneumonia and lung abscess from which she died, but all during her illness she was able to take food and medicine by mouth, and had very little difficulty in swallowing.

CASE NO. 11

Male, C.H.F., age forty-nine. Chief complaint: Regurgitation of food, difficulty in swallowing and cough. About three or four years ago patient felt as if a pocket had been left in the throat following a tonsillectomy. He consulted an ear, nose and throat specialist who found the throat in good condition. At this time patient occasionally regurgitated a small amount of food. Condition has gradually gotten worse, and at present he regurgitates after every meal two to three tablespoonfuls. Difficulty in swallowing started a year ago. After meals, coughs and regurgitates. For twenty-seven years patient has had severe headaches, also told at one time that he had a duodenal ulcer (1921). Has lost just a few pounds in weight. General appearance is that of good health, otherwise physical examination was negative. Blood pressure, 120/70.

X-ray examination: Shows a fairly large diverticulum that lies almost in the center line, and when fairly well filled is probably two centimeters in anteroposterior depth, and four in width from side to side, and probably six from top to bottom. It is of smooth outline, definitely posterior to the esophagus, and barium can be seen spilling into the lower portion of the esophagus from a point near its top. No other pathology in the esophagus. Urine: Negative. Blood: Hemoglobin, 80 percent; red blood cells, 4,185,000; leukocytes, 11,000; polys, 73 per cent; lymphocytes, 25 per cent. Patient's general condition was excellent, and no preliminary treatment was necessary.

Operation: December 16, 1929. First stage, local anesthetic, one percent novocain. The sac was freed, and was sewed to the sterno-hyoid muscle in the upper part of the wound, so that the base of the sac was a little higher than the opening in the esophagus. Wound was closed, and a rubber tissue drain inserted at the lower angle of the wound.

Patient made a very good recovery from the first operation, and was able to swallow without any difficulty the same day. He was out of bed on the third day, taking soft food.

Eight days later, December 24, 1929, the second stage of the operation was done. The sac was freed down to the esophagus and was removed by means of a transfixation suture, and several sutures into the esophagus to reinforce the opening.

Patient recovered very nicely from the second operation. Was kept without food for forty-eight hours, given glucose and saline intravenously twice, and then was started on liquids. In a week's time he was taking soft foods without any difficulty. There was no leakage at any time from the suture line in the esophagus.

This patient was rechecked on April 2, 1930, and has had no difficulty in swallowing, nor any regurgitation, and his cough has entirely disappeared.

REFERENCES

1. Rokitansky, C., quoted by C. H. Mayo: Annals of Surg. 1923, lxxvii, 267.
2. Zenker, F.A. & Ziemssen, H., quoted by Judd, E.S.: Arch. Surg., 1920, i, 38. Oekenomides, quoted by Judd, E. S.
3. Judd, E. S.: Arch. Surg. 1928, i, 38.
4. Bevan, Arthur D.: J. A. M. A., 1921, lxxvi, 285.
5. Jackson, Chevalier: Ann. Surg., 1926, lxxxiii, 1.
6. Morley, John C.: Brit. Med. Jr., 1926, i, 981.
7. Duer, quoted by Morley; Von Hofmeister, quoted by Morley.
8. Bull, P.N.: Ann. Surg., 1926, lxxxi, 59.
9. Vinson, P. P.: J. A. M. A., 1926, lxxxv, 178.
10. Flummer, H.S.: Surg. Gynee.& Obstet., 1910, x, 519.
11. Girard, C.: Assn. Franc de Chin, 1896, x, 392.
12. Murphy, J. B.: Surg. Clin. Chicago, 1916, v, 391.
13. Mayo, C. H.: Ann. Surg., 1923, lxxvii, 267.
14. Gaub, O. C.: Surg. Gynec. & Obst., 1915, xxi, 52.
15. Lahey, F. H.: Surg. Gynec. & Obst., 1926, xliii, 359.

FURTHER STUDIES IN TREATMENT OF FOOD SENSITIZATION WITH DIGESTANTS AND CITRIC ACID

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(Read before the Association for the Study of Allergy, Portland, Oregon, July 8, 1929).

My results in the treatment of sensitization phenomena by digestants have been so gratifying that I again call attention to them.

An article on the subject before the Society a year ago, included a theory to explain sensitization which now I wish to simplify. An understanding of the theory I believe to be of value, because it fits many of the facts and has helped me to develop methods of treatment and to understand many of the phenomena which I have observed.

Briefly, the history is that whole protein gets into the blood stream and tissues of the body and is digested by an enzyme to two simpler compounds. One, only, of the two, is toxic. The toxin is further digested by a second enzyme. An addition which I now make to the theory is that there may be a third or even a fourth enzyme concerned in the digestion before all the products become nontoxic. The first enzyme I designate "P", protein enzyme, and the second "T", toxin enzyme. There also may be toxin enzyme two, "T"2, and even three, "T"3; enzyme "P" may be identical for two or more proteins—nonspecific—whereas "T" is probably specific for each toxin. It is possible also that the toxin stage of the protein may be absorbed from the alimentary canal. The first splitting of the protein is simple. The splitting of the toxins is probably in two or more steps and complicated. That enzyme "T" easily falls behind "P" in their respective tasks explains how the toxins accumulate in the body—to produce chronic effects. In has seemed to me that in chronic cases the tissues are often so full of toxin that it is impossible to free the body therefrom by any method I have yet used. The theory is presented merely for its worth, without the many observations I have to substantiate it. I believe many observations to be explained by the theory.

My idea, further, is that the protein escapes digestion in the stomach because of inadequate digestion and passes directly into the tissues as whole or split protein. Many conditions, such as colds, nervousness, fear, fright, exhaustion, worry, anger, lack of teeth, hurried eating, irregular eating, poor mastication, etc., depress digestion, probably most commonly by reducing the amounts of hydrochloric acid and pepsin. A high percentage of sensitization cases have low gastric acidities, especially in the first hour or more after eating. (See table).

A partial explanation of the low acidities in certain conditions, notably, asthma, hayfever, bronchitis and pharyngitis, is that a large amount of mucus is swallowed, which being alkaline in reaction, neutralizes acid. Probably infectious processes cause gastritis and therefore directly inhibit the production of the normal stomach digestants.

EWALD BREAKFAST FRACTIONAL TESTS

45 Min.		60 Min.		75 Min.		90 Min.		
Free	Total	Free	Total	Free	Total	Free	Total	
12	27	15	27	22	34	27	38	Average of 25 patients with sensitizations.
0	26	0	102	13	35	30	43	Enteritis.
0	5	0	12	0	15	13	24	Asthma.
—	—	39	52	47	57	51	58	Asthma.
0	16	10	24	10	24	20	29	Asthma (persistent.)
44	62	49	65	60	75	67	80	Angioneurotic edema.
—	—	—	—	30	38	24	45	Enteritis
0	22	2	23	31	54	52	81	Enteritis
4	25	33	55	40	65	61	86	Enteritis
—	—	14	22	—	—	—	—	Asthma
—	—	0	18	0	15	—	—	Neuritis—Hypertension.
—	—	—	—	0	10	—	—	Trigeminal neuralgia
0	10	8	23	22	36	38	52	Hay fever neuritis enteritis
17	32	20	35	18	35	29	38	Enteritis
21	42	42	57	46	60	47	71	Enteritis
21	47	24	40	23	40	34	51	Asthma
0	11	3	18	8	23	7	22	Migraine
0	15	10	15	0	8	0	7	Neuritis
16	29	29	47	95	155	55	85	Gastritis Flatulence

Children, aged persons, the desperately ill, and those with highly sensitive throats, I have ordinarily not submitted to the ordeal of swallowing the tube. They are given acid empirically. I have found that even persons with normal gastric acidity may be greatly benefited by administration of acid for a time.

I reported the use of hydrochloric acid, pepsin, and calcium to aid digestion and with good results in numbers of cases. The calcium may lessen permeability of the mucosa of the upper alimentary tract to whole protein. The hydrochloric acid and pepsin were to facilitate early stomach digestion of proteins. The acid unmistakably did great good. The pepsin, I believe, helped in occasional cases. I have treated a few persons in whom

acid seemed detrimental and the pepsin alone beneficial.

After hearing Dr. Stirling's paper last year, I began using phosphoric acid in place of hydrochloric—giving it in the manner I had the hydrochloric. The results from the two acids are similar. Either acid in considerable quantity, the phosphoric perhaps less than the hydrochloric, increases the acidity of the urine and reduces the power of the blood to neutralize acid.

To get away from highly acid effects on the tissues and the urine, I have lately been using an organic acid (citric). It has worked in practically all cases just as well as had hydrochloric or phosphoric acid in similar cases. At the present time I am using two ounces of citric acid to eight ounces of water and giving from one to four teaspoonfuls in a glass of water sipped with food.

I have used acid in the treatment of a large number of cases of asthma, bronchitis, eczema and flatulence (a sensitization phenomenon I believe) with splendid results. I have treated also a small number each of a great variety of conditions suspected of being due to food sensitization; the results were good in a fair percentage of the cases. I shall report a series of more or less typical cases.

Case number one had severe pruritus of the hands, feet and face. This had annoyed him for months and was gradually growing worse. I gave him phosphoric acid to take with his meals and told him to come in again in a week. At the end of the week he reported that he was no better. I had him double the dose and return in another week. This time he admitted he might be a trifle improved. I instructed him again to increase the dose of the acid. When he returned the next week he said he was well. I instructed him to keep up the use of the acid, and gradually lessen the amount, for six weeks to two months; he has had no return of the trouble.

Some of these cases respond quickly to the use of acid with their foods. Pruritus cases when taken early, and when acid is administered with foods in a sufficient quantity, usually recover promptly. The acid may have to be continued indefinitely in certain individuals. In some severe cases the acids only minimize the pruritus.

Case number two is a woman who had hives for several months. She knew the diet was the etiologic factor but, change the diet as she might, she could not get free of the hives. The use of citric acid with her food gave relief in relatively few days.

Case number three was a baby, about two years of age, with mild eczema. The mother had noticed a general "breaking out" with itching. Their family physician rightly had blamed the diet and tried special diets. The condition made no improvement. Phosphoric acid caused the rash to disappear in two or three days and there has been no return. This was months ago. The medicine was given only about one week.

The mild eczemas, early pruritus and hives ordinarily respond promptly to acid with the foods.

Case number four is a girl about seven years of age, with asthma since early childhood. The attacks generally began with a cold, and soon passed into severe asthma. She was markedly sensitized to many foods —proven by skin tests. Acid was prescribed to be taken with her food whenever she had colds. This was first given her about two years ago and the child has gotten along without asthma. They failed to give acid at start of one cold and she came near having a paroxysm. The elimination of the worst reactors was undoubtedly beneficial.

Not all children who have asthma have it from food. My results with asthma in children lead me to expect good results, however, in perhaps seventy-five to eight-five per cent or even more of the cases. Most persons who suffer with asthma following colds will usually have the asthma controlled by use of acid with food during "colds."

Case number five is a man 83 years of age, who occasionally has had extremely severe paroxysms of asthma. I saw him first in an extremely severe attack and the family was of the opinion that he would not live; it even seemed to me that he might not survive. I gave him the usual emergency treatment for two or three days and prescribed phosphoric acid. The result was most satisfactory.

I could report a large series of cases of asthma in which the only, or the main, cause is food, and where the asthma has not existed for a great length of time or where the paroxysms are not frequent, I expect good results. I make it a practice to prescribe the acid when I first take charge of a case and to make the sensitization tests afterward if relief is not obtainable.

Case number six is a five-year-old boy who had bronchitis most of the time since birth until I gave him citric acid with his food, about three months ago. Before taking the acid the child practically never had been free of cough. The mother says the cough had been most annoying and frequent and

since beginning the acid he has had no cough.

Children, and many adults, who develop bronchitis with colds commonly have their bronchitis clear up promptly, I find, when given acid with their food.

Case number seven is a woman who complained of gas in the stomach and intestines which seem to press the heart, embarrassing her breathing. This patient also had asthma; it was persistent and chronic. Citric acid relieved the flatulence but it only slightly improved the asthma. Testing her for her sensitization reactions and limiting the diet have given her further improvement in her asthma.

Case number eight is a man 54 years of age, who had a great amount of stomach and intestinal distress which had been variously diagnosed and treated—uniformly without improvement. He was given acid with much relief.

There is a condition which I usually term enteritis, the symptoms of which may be described as burning, indefinite pain, discomfort, etc., in the abdomen often associated with flatulence which yields readily to the use of acid with foods.

Cases of flatulence are not as tragic as are many cases of asthma, but, nevertheless, these patients are most appreciative of relief. The administration of acid works in a large percentage—well over ninety per cent I believe—of these cases.

Case number nine was angioneurotic edema of face and eyes in a boy 21 years of age. He was practically blind. He could count his fingers when held close to his eyes with a bright light directly opposite. He suspected his condition was from food and he obtained most wonderful results from advice as to how to eat. The advice was to eat slowly, regularly, and masticate thoroughly and not to eat too much of any one thing. Within four days he came to the office without anyone to lead him. The administration of acid further improved his vision in a short time from 3/200 to 20/100 and it has been improving gradually since. I found it necessary, however, to test him for his reaction to foods and to eliminate all the reactors. This patient had been in the hands of a friend of mine in St. Louis who had recognized the condition as one from food and had tried out various diets—but without more than temporary relief. I believe the acid was beneficial in this patient although it is not easy to evaluate the results. His stomach acid was normal after an Ewald breakfast.

Case number ten is a young man 20 years of age, with migraine of two years' duration; his mother had suspected relationship between his headaches and stomach. Associated with headaches was an inflammation of the eyees. The pain in the head usually came for only a few hours at a time, but practically daily. He was given citric acid with his meals and headaches since have almost ceased. This is a fairly recent case and will have to be reported more in detail at a later date. I may have to test him for reacting foods and eliminate them. There is promise of result without this.

Cace number eleven is a young woman with fairly typical trigeminal neuralgia. She has been given acid with her food apparently with most definite improvement.

I reported previously two other cases of facial neuralgia in which, I believed, I had obtained definite proof of sensitization from food, and had benefited by improving their digestions.

The relative dosage of the acids may be figured by computing the replaceable hydrogen in each. Dilute hydrochloric acid for each 4 c.c. has .0101 gm. replaceable hydrogen. Dilute phosphoric has .0123 gm. replaceable hydrogen. As the citirc is an organic acid, hydrogen ions dissociate less than in inorganic acids. Citric acid also undergoes digestion, hence, it must be given in relatively large amounts. I find one to two grams of citirc acid to be ordinarily beneficial. The citric is less disagreeable than either hydrochloric or phosphoric acid. Certain patients refuse to take hydrochloric acid. Adding the citric acid to lemon, orange or grape fruit juice makes a palatable drink and its value is also enhanced by the citric fruit juices.

CONCLUSIONS

1. The theory I advance will help one to understand why digestants are beneficial in the treatment of food sensitizations.

2. Digestants given with food relieve many cases of various types of food sensitization—sometimes when moderately severe.

3. Citric acid has been the digestant of choice, in a large percentage of cases, in my recent work, because of its not unpleasant taste and its anti-acid effects on the tissues and urine.

4. Pepsin may be added to the acid—perhaps advantageously. Pepsin alone may be of service in occasional cases.

5. Sensitization cases ordinarily have too little gastric acid.

6. Acid is given beneficially to certain patients who have normal or practically nor-

mal acid. In such instances it is wise to reduce the citric acid when the symptoms are gone.

7. The dosage of the citric acid has been one to two grams with each meal. If this amount does not give results, larger amounts should be given. The guide, it seems, should be the disappearance of symptoms being treated or the development of symptoms from the acid.

8. Severe cases of sensitization will usually need to have eliminated all severely reacting foods, as well as taking the acid.

REVIEW OF HEAD INJURIES OBSERVED IN THE CITY COUNTY HOSPITAL OF EL PASO, DURING THE YEARS 1926 to 1929 INCLUSIVE

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The purpose of this paper, as indicated in the title, is to review the cases of head injuries seen in this hospital for the past several years, in an effort to evaluate the present methods of treatment as compared to the course followed in years past.

This paper does not include any original work, nor does it propose to present even the well known text book discussion of injuries to the skull and its contents; but deals rather with the various methods of therapy employed in this hospital, in an effort to ascertain which method of treatment appears to give the best results.

This series of 66 cases seen in the past four years in the City-County Hospital reveals a 51.1 per cent mortality for the entire period. The average age of the fatalities was 31.8 years. The average age of the cures was 16.8 years. The oldest patient that recovered was 47 years of age and the majority of the cures were under the age of 20 years. Thus substantiating in our own experience the well known fact that the prognosis is much better in the younger individual. It was found that the average period of hospitalization for the whole four years was 7.1 days in the cases which recovered.

In the year 1926 there were four cases with a mortality of 75 per cent. The average age of the fatalities was 23 years, and the average time in the hospital was 4.8 hours. The treatment at this time, as evidenced by orders on the charts, consisted principally of the treatment of shock; i.e. hypodermoclysis, morphine, application of heat to the body, and, in some cases, adrenalin.

In the year 1927 there were 13 cases, with a mortality of 61.5 per cent. The average age of the fatal cases was 28.2 years, and the average time that these cases were in the hospital was 19.3 hours. The treatment at this time was much the same as in the year 1926. But in most cases the head of the bed was elevated and an ice cap was applied to the head.

The cases seen during the year 1928 resulted in a mortality of 60 per cent. The average age of the fatal cases during the year was 34.8 years, and the average stay in the hospital was 19.4 hours. The treatment given the majority of cases consisted in elevation of the head of the bed, ice cap to the head, proctoclysis of glucose and soda and an occasional spinal puncture which was not repeated.

During the year 1929, there were 26 cases observed, with a mortality of 34.6 per cent. In the fatal cases the average age was 41.2 years and the average period of hospitalization was 25.2 hours. During this time, the treatment employed may well be divided into two parts, namely:

1. The treatment of shock. The patient was put to bed, local heat applied to the body, morphine used if indicated and a close watch kept on pulse rate and blood pressure. As soon as there was evidence of recovery from shock,

2. Active dehydration was employed; namely, repeated large doses of magnesium sulphate; glucose (50%) intravenously and spinal puncture done early in the treatment and repeated every six to eight hours as long as there was any evidence of increased intracranial tension. The average stay in the hospital in these cases which recovered was 5.8 days.

In the treatment of fractures of the skull with brain injury no definite rules can apply to all cases; however, the treatment can be standardized and from standardization of treatment and principles, certain rules can be formulated that will act as a guide in the treatment of all cases.

The conservative method of treatment has always been given preference in this hospital, rather than the more radical procedure of decompression. Decompression, however, has a place in the treatment of these cases and there are definite indications for its use; and it should be used when indicated.

The plan of conservatism which we have attempted to follow has been, first, the treatment of shock. Of course some cases

come into the hospital relatively late after their injury, and the stage of shock has passed. However, in all cases presenting evidence of shock all efforts were directed toward preventing further trauma and restoring the patient to the normal by the present accepted methods of treating shock.

On recovery from shock the treatment of the cerebrocranial injury should be instituted; i.e.

1. Spinal puncture with the removal of spinal fluid. This is repeated every 6 to 8 hours.

2. Large doses of magnesium sulphate, either by mouth or by rectum given every four to eight hours.

3. The intravenous injection of a hypertonic solution to increase the blood volume and to dehydrate the tissues. Glucose appears to be the ideal agent to use for this purpose because of its low toxicity.

In conclusion we may summarize as follows:

1. The prognosis in cases of cerebrocranial injury is much better in the younger individual than it is in the old, regardless of the type of treatment employed.

2. During the past four years the period of hospitalization of these cases in the El Paso City-County Hospital, has been reduced from an average of 7.1 to 5.8 days, in the non-fatal cases. The period of hospitalization of the fatal cases has shown an increase from an average of 4.8 hours to 25.2 hours.

3. The average age of the fatal cases has shown a steady yearly increase from 23 years in 1926 to 41 years in 1929.

4. The mortality rate over the period of four years has shown a marked decrease from 75 in 1926, 61.5 in 1927; 60 in 1928 to 34.6 in 1929.

5. Since the greatest decrease in mortality rate was coincident with the institution of a very definite plan of treatment; we feel justified in assuming that, at present, this method gives our patients their greatest chance for recovery and decreases the cost of hospitalization to a minimum.

DISCUSSION

DR. MILLER complimented Drs. Hardy, Guynes and Blanchard highly on the type of paper presented and said that the data given showed a careful observation and study by these men and was the sort of work that benefited both the doctors preparing the paper, and those who heard or read the data therein. He pointed out that the use of the term "concussion" is not an entirely satisfactory term, but that it is commonly used to imply an injury to the brain when the x-ray shows no bone injury. Frequently these cases will show intracranial injury, with spinal tap showing increased pressure and blood in the spinal fluid. He said that in re-examination of a number of old skull fractures, he was surprised to find that in these flat bones, union was by fibrous formation. Dr. Goodwin explained to me that this was the case in all thin flat bone displacement. Fracture of the skull is not necessarily a serious injury, and in many cases we doubt that there is much intracranial damage. However, in industrial cases, the presence of a simple fracture in the skull made evident by the x-ray, is what the lawyers look at, and not the absence of neurological symptoms.

DR. GOODWIN answered that fractures of the skull like fractures of the pelvis, resulted not so much in injury to the bones themselves as to the tissue inside.

DR. CUMMINS commented on the lack of follow up of these cases after they leave the institution. He asked just how much value Dr. Hardy placed on the manometer reading when testing spinal fluid pressure and whether he repeated spinal taps because of increased pressure, as shown by the manometer or upon the clinical symptoms of the pressure. He mentioned that in a recent meeting discussion arose as to the value of the manometer and that the concensus of opinion was that the manometer readings were not a true indication of increased cerebral pressure. Dr. Cummins said that he thought that the reading and discussing of papers of this sort was one of the most important services rendered to the hospital, being based as in this instance, on the work actually done in the institution.

DR. MILLER wanted to know just what the relationship was, between the pulse rate and the blood pressure which indicated the necessity for decompression.

DR. STEVENSON remarked that he thought skull injuries ought to be classified, in such manner that a better indication of the mortality rate could be obtained. For instance he described the eggshell type of injury dying within a few hours after hospitalization, and a flat depression relieved by decompression. Showing the precentage of mortality of the various types of skull injuries would be a truer indication of treatment rendered.

DR. HARDY thanked the staff for the reception of his paper and stated that he took very little credit for its preparation, and said the appreciation was really due the interns, Drs. Gynes and Blanchard.

In closing Dr. Hardy said "In all cases we have had diagnostic spinal tap. In cases which showed a high degree of intracranial tension and clinical symptoms, the spinal top has been repeated, checking every six or eight hours with the clinical symptoms. The method we have used is observation, constant observation, watching the pulse, state of consciousness, loss of reflexes, involuntary micturition and defecation; when these things occur, we know the patient is going to the bad. Restlessness is to be watched for since it points, as Dr. Dandy says, to one of two things; either the patient is coming out of it, or going deeper into unconsciousness. Occasionally we have had a case showing intracranial tension, the projectile escape of spinal fluid, not benefitted by spinal tap. In these cases decompression was done, and done quickly. There is another type of skull injury which deserves mention. The type which on spinal tap brings forth very little fluid. I would advise doing nothing, further than watching the pulse, observing the loss of reflexes, and progressive paralysis. Generally this type can take care of his disability without the necessity of operation. When the moment comes however, when operation is indicated, operation must be done, and done immediately. This is the general treatment of all these reported cass.

A CASE OF PARALYSIS, PROBABLY DUE TO JAMAICA GINGER.

JAMES J. GORMAN, M. D.
El Paso, Texas

(Presented at Hotel Dieu (El Paso) Staff Meeting on April 9th, 1930)

We are scarcely able to read a current newspaper that we do not see a report of a number of cases of paralysis due to Jamaica ginger. Since this case seems to be rather typical of that condition I wish to present it to you for your discussion.

Case No. N-364; male, white, age 37. Telegrapher and water service helper. Examined March 21, 1930. Chief complaint, paralysis of hands and legs.

P. I.—Had been in good health until Feb. 15th. On this date he was awakened by a chill which lasted about an hour. He had a diarrhea but no vomiting. Remained in bed that day and felt normal the following. On March 1st, he worked all day standing in water to his knees. On the second he noted a tired feeling in the legs which grew more pronounced and after about four days had a soreness in the calves of the legs. The knees became weak and he was forced to quit work after one week. Apparently no involvement above the knees. On the ninth his hands began to grow tired, followed by a stiffness and in a few days by tingling in the tips of the fingers which lasted about one week and later numbness. Was able to use his hands for about a week but during the past week had been unable to move the fingers of the right hand and those of the left only very slightly. At the onset had a dull pain in both shoulders, right ankle, and back but this disappeared after a few days. At no time has he had any severe pain in either arms or legs and there is no loss of sensation. At present is not able to use his hands and can only walk with support. For about one month prior to onset of paralysis was accustomed to drink about six to eight ounces of Jamaica ginger at least twice a week.

P. H.—Essentially negative except for scarlet fever and pneumonia in childhood. Influenza in 1918, and occasional sore throat.

F. H.—Father died at 88; mother at 74 of paralysis, and one sister of tuberculosis and one brother of rheumatic fever.

Phys. Exam.—Patient is well developed and nourished. He is scarcely able to walk and has a shuffling gait and a marked toe drop. General examination is negative except for neurological findings. There is practically complete paralysis of the extensor muscles of the leg and forearm. Is unable to move the toes and the fingers of the right hand. Very slight movement of the fingers of the left hand and the movement is most marked in the outer two fingers. No grip possible. Sensation to touch, heat and cold is normal. Patellar reflexes are diminished. Eye ground examination negative. Urine and blood examinations normal. Blood Wassermann negative. Spinal fluid examination showed cell count 3, globulin trace. Wasserman negative. Colloidal gold test 5555400000 (paretic curve).

Examination on April 18th showed definite atrophy of the small muscles of the hands. Patient felt that he was some better, stating that he seemed to have slight use of his hands. This I was unable to confirm. On the possibility that arsenic might be present sodium thiosulphate was given without apparent improvement. Through the courtesy of Drs. Ramsey, Black, and Hardy a specimen of the Jamaica ginger used by this man and another man who was under the care of Drs. Hardy and Black was obtained. The specimen was labeled as containing 83 per cent alcohol. Dr. Black and I have had specimens examined by three chemists working separatelty. So far we have obtained nothing but negative data. Examination for arsenic, lead, methyl alchohol and phenol groups are negative. Further examinations are now being made and we hope to have a more complete analysis to present at the next meeting.

REPORT OF THREE TUMORS OF THE NECK

W. L. BROWN, M. D. and C. P. BROWN, M. D.
El Paso, Texas

(Given at the Staff Meeting of Hotel Dieu, El Paso, on April 2, 1930.)

CASE 1.

Mrs. R. M. C., age 39, married. No serious sickness. For eight years has had a small lump on left side of her neck just below the angle of the jaw; for the past year it has been enlarging perceptibly; it is now about the size of a medium size hickory nut. The tumor is excessively hard, almost like cartilage; it is moveable, not involving skin or deeper tissues. It is not tender on pressure and never has been. No definite preoperative diagnosis was made. Tumor was looked upon as a gland and possibly tuberculous in nature. It was removed under local anesthesia and gas; it was shelled out and had a definite capsule. It had no connection with the parotid gland.

Postoperative diagnosis: Mixed Celled Tumor.

CASE 2.

Mr. B. G., Mexican, age 35. Was struck on right side of face in the region of parotid gland over three years ago. Soon after a tumor formed; it has been increasing in size for the past seven months; has never been painful or tender on pressure. It is fairly moveable, excessively hard and slightly nodular. No glands under his arms or elsewhere. Preoperative diagnosis in this case was mixed celled tumor. It was removed under general anesthetic and was found to be in close proximity to the parotid gland and facial nerve. It was shelled out without any injury to either.

Postoperative diagnosis: Mixed Celled Tumor.

DISCUSSION:—These mixed celled tumors are only very mildly malignant, if at all. If the capsule is ruptured in their removal, there is a possibility of their return. Ewing states "In the salivary glands, buccal, mucosa, palate, lips, neck, orbit, lacrimal gland and face occurs a group of tumors about the origin of which has long been maintained an active controversy. These tumors are of complex structure, usually presenting epithelial elements in the form of cell strands, alveoli or diffuse masses, and mesoblastic tissues, chiefly cartilage, mucous tissue, and cellular connective. Any one of these elements may predominate, giving nearly pure chondromas, sarcomas, or carcinomas, but usually all the cell types are represented." Grossly these tumors resemble one another very closely. They are round, nodular, not tender or painful, grow as a rule not in the gland but in close proximity to it. They occur very commonly in the region of the parotid, submaxillary and soft palate, and occasionally just lateral to the midline of the upper lip.

CASE 3.

Mrs. H. S., married, age 36. No serious sickness of any kind. Is not subject to tonsillitis. A few weeks ago went to Albuquerque to take chiropractic treatments on account of severe headaches. She now comes complaining of swelling at the base of

the neck on left side just above clavicle. This she noticed about five or six weeks ago. She had not been sick prior to that time; was only attracted by the swelling; no pain or soreness; neck feels tired at times; says it has not enlarged much in size since first noticed. States that it is much larger at night than in the morning.

She has a good many filled and crowned teeth; tonsils are quite normal; just over the inner end of the left clavicle, there is a swelling about 1¼ inches in diameter that extends well down under the clavicle; is quite moveable; is not tender on pressure; there are no glands about the neck or under the arms. Blood: White cells, 8,000; polys 66 per cent. Wassermann negative.

DISCUSSION:—This tumor is not as hard as either of the above; is of short duration. It was removed under general anesthetic and there were found three other small glands, closely connected with it. This finding made it certain that it was not a giant cell tumor. The laboratory report was Hodgkin's disease or possibly lymphosarcoma.

DR. ROBERTSON, Professor of Clinical Pathology, Mayo Clinic:—The subject of mixed celled tumors of the parotid is an interesting one and their classification has varied. Many years ago they were diagnosed as endotheliomata, which at that time was rather a waste paper basket diagnosis. The tendency today is to regard these tumors as a low grade malignancy of the carcinomatous type. The question of course arises as to why the presence of cartilaginous material and sarcomatous like cells. Pathologically they are really carcinomatous. They may occur any place about the cheek or mouth. They occur in tissue which is embryologically and histologically inactive. In other words, of a low grade activity histologically. Therefore, they should give rise to low grade growths. To me the presence of cartilage is accounted for by the slow growth of the connective tissue with an associated large amount of mucus and therefore the formation of cartilage.

It is not safe to give a favorable prognosis in these cases even though most of them get well after removal of the tumor, for it is well known that a percentage, though small in number, grow fast and terminate fatally.

Dr. Waite, as all good pathologists are wont to do, is certainly correct in guarding his diagnosis of Hodgkin's disease in the last case, because, as we all know, Hodgkin's disease may produce most any appearance.

CASE REPORT
(Compound Comminuted Fracture Both Bones Left Leg.)

CHARLES N. PLOUSSARD, M. D.,
Phoenix, Ariz.

(Read at monthly Staff Meeting of St. Joseph's Hospital, at Phoenix, Ariz.)

Patient, an adult white male, was brought into hospital on stretcher conscious and rational with history of having been hit by car while riding motorcycle. Immediate examination revealed left leg badly crushed, foot rotated so that toes were pointing posteriorly, markedly swollen with deformities and malposition almost beyond description. Tibia protruded through skin above ankle and again just below the knee. Limb was bleeding profusely and fat, bone marrow, fascia, and muscle extruded from wounds. Patient's general condition was fair. Morphine gr. ¼) was given immediately by hyperdermic. The leg was put up in a Thomasy extension splint with extension from foot by means of a flat piece of steel which was fastened around arch of foot and heel with adhesive. Bones moulded into best possible position (Fig. 1) but no great amount of ma-

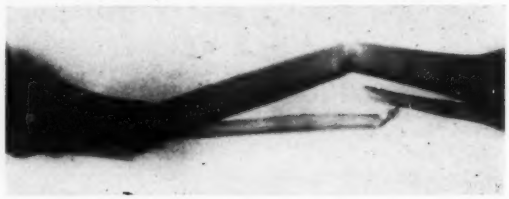

Fig. 1

nipulation was done at this time because of the extensive injury already done to soft tissues. Fifteen hundreds units antitetanic serum was given. The following day the patient's condition was improved but on the second day after injury the leg was much more swollen, temperature 104, and he was

Fig. 2

irrational. There was a large amount serosanguinos drainage requiring dressing every two hours. Odor from wounds aroused suspicion of gas bacillus infection. X-ray taken at this time showed some air in tissues but no definite crepitation could be determined. Patient's condition was not good and he

Fig. 3

appeared quite sepitc. Hot magnesium sulphate packs were applied continuously. The following day he looked even worse; abdomen was distended; limb was more swollen and more bluish in color. Drainage of bloody serum became more abundant. Patient's general condition grew worse; temperature and pulse mounted and leg looked as if amputation above knee would be necessary. Morphine was required for pain, and intravenous glucose and saline were given with patient taking large amounts of water by mouth. The next day, (five days after injury), patient's condition seemed improved. limb not being swollen so and color appeared better. On March 23rd, (ten days after injury), patient's condition was much better, the leg was much less swollen. A large fluctuating area in limb was incised under local anesthesia and a large amount of pus mixed with debris, consisting of muscle tissue and fascia, escaped. Wound was dekinized every

three hours. Bones were moulded into better position and apposition plates applied with lateral pull over angulation of fragments. More extension was also aplied to foot. The condition of the limb and general condition improved steadily. On April 27th large fluctuating areas over entire leg between fractured bone ends appeared. Four incisions were made into these areas, eminating large amount of pus and debris including, it seemed, all kinds of soft tissues: for example, nerves, muscles, tendons, vessels and fascia. Wounds were darkinized every three hours. Tibia between fractured areas was absolutely devoid of any muscle attachments. It appeared dead with surface resembling a bone from which the tissues had been removed by boiling. The following day, patient had a chill accompanied with nausea, vomiting, and inguinal adenitis. Temperature was 105.6 pulse 150. He was quite irrational. Redness, tenderness and pain extended above knee on inner side of thigh almost to scrotum. The next day his condition began to improve and cellulitis above knee gradually subsided. Condition continued to improve steadily although there was still much drainage from many sinuses in limb still present. On May 11th, there seemed to be some union at both fractured areas and a week later the Thomas splint and extension were removed and limb placed in basket splint. Drainage and swelling were gradually becoming less. Movement in ankle and knee was begun more vigorously. He was allowed up in the wheel chair and was discharged from hospital on June 5th. There were six draining sinuses still present in infected area. On several occasions, small sequestra were removed from draining sinuses. On July 24th, x-ray showed large sequestrum at lower fractured area apparently separated (Fig. 2), . Under spinal anesthesia an incision was made over anterior tibia at lower fracture. Three quite large sequestra were removed leaving a gap between fractured ends of bone about one and one-half inches wide. This was packed with iodoform gauze and left wide open. Two days later patient had a severe chill. He was not irrational but temperature went to 104 and pulse 130. Condition of patient no tbad, however. He improved rapidly after this. A posterior plaster cast was applied and patient dismissed from hospital on crutches. He has been reporting to the office about twice a week. On September 30th, diathermy three times weekly was started, with one electrode on sole of foot and the other above the knee. Improvement was noticed almost at once. Drainage lessened and condition of limb including knee and ankle joints improved with remarkable rapidity.

In January, 1930, he was walking with cane, movements at knee and ankle being almost normal, with union fairly firm in both fractures (Fig. 3). No enlargement of limb. Still two small drainage areas suggesting presence of small sequestra. These will no doubt come to the surface soon. He is able to drive a car with no inconvenience whatever and says limb feels normal in every respect.

In May, 1930, patient resumed his duties as a motorcycle policeman.

CASE PRESENTED FOR DIAGNOSIS

DR. H. S. NEWMAN

(El Paso City-County Hospital Staff Meeting, April 16 1930)

R. R. A., male, white, age 26, mechanic. Wife living and well. Family history negative. Normal diseases of childhood; influenza several years ago, and "rheumatism." Rest of history negative.

Present Illness.—Feb. 25th, he arrived from Yuma, Arizona, where he had been working as indoor mechanic for two or three months. Came by auto and camped out two or three days. Doesn't know exactly when he started being sick, but says he had been feeling ill for two or three days before leaving Yuma. Complained of general lassitude, severe headache, loss of appetite, belching, gassy and uncomfortable abdomen, little cough, and afternoon fever. He no diarrhea and said bowels were regular. There was no urinary disturbance (but I see that Dr. McCamant has written on the history sheet that he had some pain on urination, but that it was not constant). His chief complaint, then, was headache, uncomfortable abdomen, general lassitude, and fever. He had no chills at all and no sweating up to this time.

I examined him at night at one of the camps on Feb. 27th. The lighting was poor but I could make out what appeared to be a few rose spots on abdomen and chest. He was a husky, well nourished individual. Temp. 104.4, pulse 96, resp. 25, blood pressure, 117/78. Tongue was coated, mouth and throat otherwise clear. Lungs negative. Heart negative, except for a loud systolic murmur at second left interspace, sternal margin. Abdomen was distended and much rumbling around of intestines was heard. No tenderness nor rigidity. Spleen not palpable and liver not enlarged. He had a little soreness in back of his neck on flexion, but no rigidity. Reflexes were apparently normal. Examination otherwise negative. Next morning I went to see him again. His temperature was then 102.2, pulse 96; otherwise his condition was unchanged. I sent him to the hospital with a working diagnosis of typhoid. There he was shifted around from one service to another, everybody disclaiming him. He ran an irregular febrile course for 36 days in the hospital and possibly for four or five before entering, making something over forty days with fever. His morning temperature was low, usually around 100, but for the first four or five days it was around 102, the afternoon temperature was around 104 for the first six or seven days and then around 102 after that. After about his 14th or 15th day in the hospital the morning temperature touched normal or below, about the 32nd day his fever started to break and from then on was seldom over 100 in the afternoon till the 36th day when it reached normal and stayed there. His pulse during all this time never went over 100 and was usually around 90. His headache disappeared soon after entering the hospital, his appetite became good, and the only thing he complained of was fever, and severe drenching sweats nearly every night, unassociated with chills. He had one chill during his entire stay in the hospital. At one time, for several days, he complained of a little soreness in the gall-bladder region, and the upper level of the liver seemed a little high. Dr. Cummins examined him but found no surgical condition. Dr. Liddel examined for tuberculosis with negative results. An x-ray of his chest was made and Dr. Waite made the following reading: "Film shows a rather large heart shadow and the aorta appears to be wider than normal. There are some old scars in the region of each hilus, otherwise there are no marked changes. This is suggestive of a syphilitic heart disease." His white cell coust was 14,000 with 83 per cent polys; no parasites were found in a blood smear; his blood Wassermann was negative; Widal at first was reported negative and then positive and again negative. A rechneck at one of the uptown laboratories proved to be negative. A blood culture at first was negative but another on the 12th day was reported positive with "active, gram negative bacilli, resembling typhoid." Neither of these cultures though, were incubated for more than three

days. Two agglutination tests for Br. abortus and melitensis were negative in a dilution of 1 to 200. A lower titer than this was not tried for some reason. On the 6th day a blood sugar of 140 mgm. was reported. On the 17th day his white count was 12,000 with 68 per cent polys. His urine on several occasions was reported negative except for "few" or "occasional" pus cells. A stool culture on the 29th day was negative for typhoid bacillus.

The point of interest in this case is that this man was in the hospital for 30 days, was seen and examined by the internes and by several of the staff members, including myself, a good deal of laboratory work being done, and still undiagnosed. Dr. McCamant finally stepped in on the 30th day of his stay in the hospital and supplied us with diagnosis. Otherwise he would have been discharged undiagnosed. His diagnosis was that of pyelitis; whether or not it is the correct one, I do not know. At least I cannot disprove it. On the 31st day he started giving him two capsules of caprokol three times per day and on the next day, after taking only capsules, his fever started to break, but it had been showing signs of wanting to break before then, I think. I might add that the patient had been given a course of quinine with no effect whatever on his temperature curve; also that the systolic heart murmur heard on the first examination showed no variation throughout the illness and that no petechiae were ever observed.

DISCUSSION

DR. McCAMANT spoke of the contradictory symptoms of this patient, called attention to the fact of his having one chill followed by temperature of 104.5 and after this temperature severe sweat. His blood count was relatively high. No tenderness anywhere in the abdomen except between the gall bladder and McBurney's point and no pain even at that point. The patient's temperature ran along at 99 and 98. Widal five or six days after entrance (having been sick five or six days before coming to the hospital) was negative, but was positive on the 14th day. He had had typhoid inoculation in Arkansas which might have accounted for the positive Widal. The second time the man was sent to my service, I saw him following one of these heavy severe sweats, and he complained of some burning on urination. There was no prostatic infection. He had some internal hemorrhoids, but not sufficient to give him the trouble that he gave evidence of. He continued to have night sweats, and to run an intermittent temperature. He was then put on caprokol and the sweats stopped, and his temperature subsided. I claim the man had pyelitis. Urinalyses showed pus in small amounts.

DR. NEWMAN replied that the man's temperature and sweats had somewhat subsided before he was given caprokol and that he was of the opinion that the temperature course was about over, and would have remained down without further treatment.

The question arose as to whether or not there was an endocarditis. Dr. McCamant said the main thing agains that was that the man got well. Dr. Gallagher replied that the man had had it once and got well, why not again?

DR. CUMMINS told of examining the patient while under surgical service. He said that he could find nothing indicative of trouble with the gall bladder, no tenderness anywhere in the abdomen, and nothing to show that he had much the matter with him in the genito-urinary tract. He stated however, that in his opinion the man should have had a cystoscopic examination and urine culture.

DR. GORMAN made the following summary, "I think this case demonstrates the necssity of accurate laboratory data. Such conflicting data as we have in this case makes it difficult of interpretation. It occurs to me that if we had a kidney infection sufficient to give the constitutional symptoms recorded and the blockage which would be expected from the urinalysis that we would find tenderness over the kidney region. Another point is that when the temperature subsided we would certainly expect to find more than an occassional pus cell present. The relatively low blood count with such a high degree of fever is against pyelitis. There is an old endcarditis present. A subacute endocarditis is not necessarily fatal and in my observation blood cultures will not be positive in more than 40 to 60 per cent of the cases. In my opinion this man had a subacute endocarditis."

DR. JAMIESON: "I quite agree with Dr. Gorman that if this were primarily a case of pyelitis, there should have been more than just a few pus cells. The patient would have had tenderness over the kidney if the infection was located there. There is another possibility and that is that he might have had an old infection of the posterior urethra, dropping out occasional pus cells. The possibility of gonorrhea is ever present. In my opinion, subacute endocarditis, appears to be the most likely diagnosis."

DIAGNOSTIC FORUM

DIARRHEA AND PAIN IN THE RIGHT FOOT

(Case 15201, Case Records of Massachusetts General Hospital, from New England Journal of Medicine, May 16, 1929).

CASE RECORD

A married American woman forty-seven years old entered April 30 complaining of intermittent diarrhea of four months' duration and pain in the right foot and leg for a week.

Five months before admission she had an attack of grippe. She stayed in bed a week, then felt well until the end of the month. Then she had an attack of diarrhea lasting a day with some fever and cramplike generalized abdominal pains. After this she had constipation for four days, the pains continuing. She strained a good deal at stool and developed hemorrhoids. She took laxatives until her bowels began to move. Her physician gave her some medicine which seemed to keep her bowels regular. The pains persisted for two weeks. During that time she stayed in bed. After this attack she had six or seven similar ones, each a little longer and more severe than the last, always accompanied by the pains and followed by constipation. She had not however been forced to go to bed since the first attack until the last one. The stools had been soft, but otherwise normal. February 28 she was examined at the Consultation Clinic of this hospital. Vaginal examination was negative. The urine was negative. There were some small hemorrhoids. Proctoscopic examination showed a pale, slightly granular mucosa but no ulcerations. The whole right side of the abdomen showed tenderness. Fluoroscopic examination with a barium enema showed that the whole colon filled normally. The tip of the cecum was displaced toward the midline and overlay a loop of the sigmoid from which it could not be separated. The colon seemed somewhat limited in motion. Chest examination showed the diaphragm in the normal position. The lungs were clear. A diet was advised which the patient did not follow. Four weeks before her admission to the hospital the last attack of diar-

rhea began suddenly. Her bowels moved three or four times a day at first, then one day moved fifteen times. The condition, including the usual cramp-like pains, became gradually worse. For two weeks she had had attacks of nausea without relation to eating and had vomited several times. For a week she had had difficulty with micturition. Then she began to have severe dull aching pain in her right knee radiating down the leg to the foot and most severe in the great toe. The leg and foot became cold and tingled. This condition became gradually more severe. Pills given by her physician relieved the pain, diarrhea and cramps. Her bowels had not moved for four days before admission. Three days before admission she found the foot was becoming purplish, especially in the region of the great toe. The color gradually deepened. She had been in bed for four weeks. During this attack and the previous ones she had had fever, 101° to 102°. She thought she had gained about two pounds since the onset of the illness.

One brother died of tuberculosis. The patient was not exposed.

She had always been well and strong except for an attack of influenza twelve years before admission. She was in bed for two weeks. She had had one miscarriage following a fall. Her bowels had been slightly constipated for years.

The patient was having such severe pain in the leg and foot that the history was left unfinished.

Clinical examination showed a fairly well developed and nourished woman lying in bed groaning. Skin hot, dry, very coarse. Half dry. Marked pyorrhea. Tongue showed a wavy brownish white coat. Boggy submental glands. Abdomen distended but rather soft, tympanitic. No dullness in the flanks (?) palpation difficult. No organs or masses felt. Liver dullness at the fourth rib anteriorly. Vaginal examination showed creamy discharge; tenderness in the left lower quadrant on bi-manual palpation. Rectal examination moderately painful; nothing was felt. Right ankle cool, foot cold and tender. On the right great toe was a circumscribed deep purple area, tender, not swollen. On the heel was a similar patch. No dorsalis pedis pulsation was felt. Pupils contracted (morphia); reactions normal. Knee jerks normal.

Before operation urine normal in amount, specific gravity 1.008 to 1.012, a few leukocytes and rare red blood corpuscles at two of three sediment examinations, 50 to 60 leukocytes per field at a fourth. (No catheter specimen.) Blood before operation: 19,000 to 9,000 leukocytes, 84 per cent polymorphonuclears, hemoglobin 75 per cent, reds 5,350,000, slight achromia. Two Hintons negative. Stools: guaiac negative at six examinations.

Before operation temperature 99° to 103.5° with two drops to 97° and 97.6° May 9 and 11.

At x-ray examination the arteries of the right leg were not visible. The bones showed no definite variation from the normal. The lung fields were clear. The diaphragm was rather high on both sides. The heart shadow was not remarkable,—possibly a little prominent in the region of the left ventricle.

With enemas the distention became less and the bowels began to move normally. The patient continued to have considerable pain in the right foot. May 19 the right leg was amputated at midnight under spinal novol. Pathologic examination of the popliteal, anterior tibial and dorsalic pedis arteries showed thickening of the intima and an increase of fibrous tissue in the media; no calcification.

May 24 the patient vomited. Four days later she was still vomiting, had a chill and very poor pulse, and looked quite ill.

May 29 a second operation was done.

June 9 there was some sloughing of the fascia from the leg wound. Two days later there was more separation and more sloughing. There was only occasional vomiting.

June 12 Alpine lamp treatment was started. The separation and sloughing increased. A large decubitus developed over the sacrum. The patient became irrational. She took little by mouth.

June 17 she died.

The following discussions of this case were presented before the Clinical Club of Phoenix, at their regular Monday luncheon meeting, Jan. 8, 1930:

DR. F. C. JORDAN

The salient points in this history are:

First, intermittent attacks of diarrhea and constipation, always accompanied by cramps and fever; each attack seemed a little longer than the previous one.

Second, severe pain in the right leg and foot, obstruction of the arterial supply terminating in gangrene and a mid-thigh amputation of the right leg.

Her past history was, essentially, negative.

Clinical examination: A married woman, aged 47, fairly well nourished, groaning with pain, some fever, marked pyorrhea. Chest, clear; abdomen, soft and tympanitic, no masses felt, no dullness in the flanks. Vaginal examination negative except for a creamy discharge. Right ankle cool, foot cool and tender with a deep purplish circumscribed area on the great toe which was tender but not swollen, and a similar patch on the heel. The urine was essentially negative except for a fairly well fixed specific gravity of 1008 to 1012. There was a constant leukocytosis with a high percentage of polys. What was the cause of the intestinal symptoms? There was some attachment of the cecum to the sigmoid which could not be separated under x-ray manipulation. Adhesions might cause a partial intestinal obstruction with all the above symptoms. Was there a malignant growth in the lower intestinal tract? The perfect filling of the bowel with barium, the absence of loss of weight and the absence of marked anemia would make this diagnosis improbable. We believe the alternating constipation and diarrhea were caused by adhesions which produced a partial intestinal obstruction. What was the cause of a gangrene of the right foot? The causes of gangrene are thrombo-angiitis obliterans, Reynaud's disease and arteriosclerotic changes We do not believe it is thrombo-angiitis obliterans for the following reason: It is almost invariably found in men; it is most common between the ages of 20 and 40 and the examination of the arteries of the amputated leg were not characteristic of this disease. We would rule out Reynaud's disease because it is nearly always symmetrical and there are practically no pathologic changes in the blood vessels How about arteriosclerotic gangrene? This is usually found in older people. There is a high blood pressure and palpable sclerotic vessels. The absence of these characteristics would be against this diagnosis, yet, we believe this is the most probable cause of the gangrene.

Our diagnosis is death was caused by sepsis following amputation of the leg. The gastro-intestinal symptoms were due to a partial intestinal obstruction due to adhesions.

DR. FRANK J. MILLOY

An analysis of the symptoms and physical findings in this case fail to reveal any pathognomonic sign or finding by which we can associate this patient's diarrhea with the pain in the right foot. The pain in the right foot and the subsequent history of this symptom is a clinical entity. From all the negative examinations of the colon and the stools, the most likely explanation of the diarrhea is an irritable colon. However, there are several causes of intermittent diarrhea which have not been eliminated in the examination. A primary carcinoma of the colon might cause diarrhea but it would have been found, no doubt, by barium enema and the guiaic examinations would have been positive. An infestation by amebae or other intestinal parasites could cause such a diarrhea but the history is not very typical and apparently this was not suspected or a more thorough stool examination would have been made. A latent syphilitic infection will cause diarrhea and really syphilis has not been eliminated in this case by two negative Hintons. However, the most plausible explanation of the diarrhea such as this which is characterized by intermittent spells of cramps and constipation is most likely an irritable colon which was a complication of the attack of influenza which just preceded the first spell of diarrhea.

The causes of gangrene of an extremity are: diabetes, Raynaud's disease and thrombo-angiitis obliterans or so-called Buerger's disease. Diabetes seems to be eliminated by the low specific gravity of the urine although nothing is said of urine or blood sugar. Raynaud's disease is a condition also known as symmetrical asphyxia or gangrene and is a vasomotor or trophic neurosis. It is most usually observed in neuropathic or emotionally unstable individuals or patients who have suffered from long standing constitutional disease. It is characterized by coldness and discoloration of the extremity and often develops dry gangrene. There is no demonstrable anatomical change in the blood vessels. During the progressive stage of the disease, it is usually painless and when recovery commences there may be more or less severe tingling and burning pains in the extremity. The majority of cases terminate in recovery.

The sudden onset of severe dull aching pain in the right knee radiating down into the leg and foot, the purplish discoloration especially in the region of the great toe, the cold tender foot and ankle, the absence of swelling and the absence of pulsation in the dorsalis pedis and the rapidly progressive course of the disease is a classical description of thrombo-angiitis obliterans. The pathological examination of the arteries of the leg describes the periarteritis which occurs in the healed or latent stage of this condition. The acute stage of this patient's condition apparently passed without any noticeable symptoms. It has probably extended beyond the arteries of the thigh to account for the advancing gangrene. The post mortem may show a thrombosis in the acute stage of this condition in some of the pelvic arteries. The woman must have died from sepsis from the gangrene extending beyond the thigh and the decubitus over the sacrum. There seems to be no way to associate the obliterative thrombo-angiitis with the preliminary history of diarrhea.

Diagnostic conclusions by members of the Club on this case were as follows:

Dr. V. Randolph: Pelvic infection; thrombosis of right iliac artery.

Dr. A. C. Kingsley: Infection in right pelvis; arterial thrombosis in pelvis.

Dr. R. J. Stroud: Endarteritis obliterans; infection right pelvis, with pressure or obliteration right iliac artery; carcinoma right pelvis; death from sepsis.

Dr. H. Randolph: Pelvic infection involving colon also; thrombosis femoral artery.

Dr. T. T. Clohessy: Embolism from some abdominal condition.

Dr. S. I. Bloomhardt: Thromboarteritis obliterans; pelvic infection.

Dr. Fred Holmes: Raynaud's disease or thrombo-angiitis obliterans.

Dr. J. M. Greer: Thrombo-angiitis obliterans.

Dr. W. W. Watkins: History suggests malignancy of bowel, malignancy in pelvis, infection in pelvis; clinical examination suggests malignancy or infection in pelvis, thrombosis of iliac artery; laboratory findings suggest infection in pelvis and thrombosis in right iliac artery.

The following discussions of the case were given by Group II, before the Yavapai County Medical Society and Medical Officers of Fort Whipple, at their meeting of February 11, 1930:

DR. W. C. MELOY

We are presented with two symptoms, which may be coincidental or may each be etiological factors. The first is diarrhea and pain followed by constipation. The causes for this are very numerous, many of which we feel we can exclude without going into. Simple enteritis would hardly have gone on to this violent ending. A more progressive enteritis would certainly have shown blood in the stool, but all guaiac tests are negative. There may have been syphilis, tertiary form, which could give pains, diarrhea and vomiting, but both Hintons are negative. There could be nerve irritation following influenza which could give spasm and hyper-peristalsis and consequent diarrhea. It could be a spasmodic condition causing an ischemia with the same results, or it might go on to a type resembling Buerger's disease, which would give us thrombi throughout the mesentery. However, the laboratory report gives us a high leukocytosis, with 84 per cent polys; the temperature has been high and to my mind, this woman presents a picture of infection.

The cecum is well over towards the midline and is apparently bound down to a loop of the sigmoid. This could be caused by perityphlitis or by any inflammatory condition of the pelvis. It is very suggestive that it has something to do with the generative organs because of the purulent discharge which was noted later and the pain in the pelvis.

The second condition: We are sure there is an arteritis but we do not believe that the arteritis is sufficient to cause the gangrene that we see presented. There is absolutely no history of intermittent claudication or any symptoms that would point to poor blood supply up to one month before the patient was taken in this last illness. It should not be a generalized arteriosclerotic condition because in this late stage the x-ray would have shown it and the symptoms should have been gradual. It is not one

of the nerve lesions because the intima is definitely thickened and the media definitely fibrotic. It must have been a chronic endarteritis because fibrous tissue is not an acute formation. With a luetic condition we would expect more sclerosis and the same thing in a diabetic condition. We are not given any definite report as to sugar in the urine but the low specific gravity would lead us to rule diabetes out.

Buerger's disease, in the moderately advanced form, could very readily give this picture, but from the pathological examination, no thrombi were noted in the area examined, so evidently the stopping of the arterial flow was well above the mid-thigh. The gangerne at first was dry. The foot was cold and edema was noted following the first operation. No mention is made as to whether or not the stump bled freely. We are led to suppose so or they would have amputated still higher. Then there is sloughing of the wound, which may be due to infection or to wet gangrene.

A mass in the right side of the pelvis could be either inflammatory or expansile, and as it expanded could press upon the iliac artery sufficiently to slow down the blood supply to the already damaged femoral artery to such an extent that thrombi could form in the iliac or femoral arteries very easily. Adhesions in the right side of the pelvis, by contracting, could cause the same results. The pressure could go on to such extent that the veins would be contracted down and cause wet gangrene. In this case, however, it would be necessary for collateral circulation to have taken the blood into the leg.

The patient's vomiting and the general septic condition, to my mind, points very strongly to infective process in the pelvis associated with a definite chronic endarteritis.

DR. JOHN D. BROOKS

My colleague has covered this case pretty well and there is very little left for me to say.

It is self-evident from the history as given us that this woman was suffering from a condition of some obstruction to the circulation in the lower extremity. I don't say that that was all, but the fact that the leg was amputated in mid-thigh shows that it was very grave. Results of that amputation, as given by the pathological examination, show obstruction in the vessels, the vessels are narrow, but they say nothing of any thrombi being found, so that the thrombi must have been higher up. I believe this woman had thrombosis up above.

It does look like Buerger's disease except, as Dr. Meloy has told you, it was very slow in coming on. She had other preliminary symptoms such as nausea, vomiting, cramps and diarrhea. I am inclined to attribute that to some intra-pelvic condition, of which nothing is given us except that one examination showed the cecum over toward the midline and overlying a loop of the sigmoid and attached to it. That could come from almost any inflammatory condition there. What diarrhea and cramps had to do with the thrombus, or blocking of the artery of the leg, is not at all clear.

Evidently they did not go high enough at the first operation because there was sloughing of tissues and true gangrene. Undoubtedly she had on infection which became very much worse—all her symptoms became worse. They tried to clean it up with the alpine lamp but it became worse.

What the pathology is in the pelvis I am at a loss to say. I believe that there may be inflammatory process, infection even, in the pelvis. I also think it possible that she had minute thrombi in the mesentery prior to the occlusion of large vessels in the leg that may have given her cramps and diarrhea. What the origin was, I am at a loss to say, but there was something pressing there. If the temperature had been a little more continuous in the beginning, it would have looked like inflammation of the bowel, such as typhoid, but that is ruled out because of the negative stools. With diarrhea for months and hemorrhoids, it seems strange to me that there was no blood in the stools.

Our diagnosis is occlusion of the blood vessels of the leg, amputation, re-amputation, sepsis and death.

The following discussions were given at the Clinico-Pathological Conference at the Messachusetts General Hospital (see above journal).

DR. RICHARD C. CABOT

They are looking hard for evidence of intestinal obstruction or intestinal ulceration, but they are not finding it, and my guess is that they went on to physical examination somewhat baffled.

We do not yet know how many examinations of the urine there were, or whether these figures constitute any limitation in the swing of gravity.

Summary:—Here is a gangrene, I suppose arteriosclerotic in origin, although the woman is young for it, with no embolic source indicated in the rest of the body. We shall have to speculate later about the abdominal symptoms.

The second operation was I assume on the same leg, but I do not know.

DIFFERENTIAL DIAGNOSIS

The striking thing about the patient in the first place is that she is so young. Forty-seven is young for a gangrene which we must, so far as I see, call arteriosclerotic. With the same symptoms occurring at seventy-seven or sixty-seven we should say, "Of course, that is what we expect." But at forty-seven it is unusual. Nevertheless I do not see evidence for any other cause.

What about the abdominal symptoms? Naturally, with a proved vascular trouble in the leg one speculates as to whether there is a similar vascular trouble in the abdominal arteries. We know something about cardiac arteriosclerosis, something about arteriosclerosis in the extremities. We have never, so far as I know, been able to construct a satisfactory clinical picture of abdominal arteriosclerosis. And yet the pathologists often point out to us cases in which there is more extensive arteriosclerosis in the abdominal vessels than anywhere else. It seems as if there ought to be a well outlined clinical picture of abdominal arteriosclerosis, but I think it still has to be said that there is not. I have seen quite a number of cases, and read of others, with about as much as this and about as little, in which extensive changes were found in the abdominal arteries. I do not know any better reason for her alternating diarrhea and constipation.

PRE-OPERATIVE DIAGNOSIS

Abdominal malignancy.

SECOND OPERATION, MAY 29

Under local novocain a left rectus incision was made. The peritoneum was found to be half an inch thick. It was opened with some difficulty. Every portion of the viscera that could be exposed was studded with myriads of small white opaque areas which had more the appearance of tuberculosis than of carcinoma. There was very little fluid. Some of the adhesions were indurated. Some sections were obtained for pathological examination. The wound was then closed.

PATHOLOGIC REPORT

A fragment of fat from the abdominal cavity showing on microscopic examination the histologic structure of caseous tuberculosis.

FURTHER DISCUSSION

Dr. Cabot: Tuberculous peritonitis—I did not in the least suspect that. I do not know now how much it had to do with the death. I judge they did nothing about it at the operation. There was nothing particular to do. Abdominal tuberculosis does not ordinarily give rise either to diarrhea or to intestinal obstruction. One makes the diagnosis when there is ascites or when there are palpable masses, and not otherwise. We did not have any of those, so I do not think we could have been expected to make it.

What are we to anticipate postmortem aside from the arteriosclerosis which we know exists at the periphery and may assume exists elsewhere? I have no means of prophesying anything else. Of course it is a good guess that she had a bronchopneumonia, but there is no basis for that except her general condition and the commonness of bronchopneumonia near death.

A Student: How about Buerger's disease?

Dr. Cabot: I do not see any evidence of that because we have nothing in the other foot and because the case is not a chronic one. Unilateral disease is not common; and she is not of the race in which that ordinarily comes.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD).

Tuberculosis of peritoneum.
Gangrene of right leg and foot.

DR. RICHARD C. CABOT'S DIAGNOSIS

Arteriosclerosis.
Bronchopneumonia?

DR. TRACY B. MALLORY

ANATOMIC DIAGNOSIS

1. Primary disease.
   Tuberculous peritonitis.
   Tuberculous salpingitis.
2. Secondary or terminal lesions.
   Pulmonary embolism.
   Bronchopneumonia.
   Thrombosis of right iliac artery and vein.
3. Historic landmarks.
   Amputation of right leg.

The postmortem findings, I am afraid, will do very little to explain the clinical picture. We found a considerable amount that was not forecasted clinically, and no explanation for many of her symptoms.

She had a very extensive tuberculous peritonitis of the dry, adhesive type. This apparently arose from a tuberculosis of the Fallopian tubes, one of the common sources of tuberculous peritonitis in women. There was a chronic endometritis which may possibly have been tuberculous, although the histologic picture was not entirely characteristic. She did have a slight terminal bronchopneumonia. It was not tuberculous. There was not much tuberculosis in the lungs and none of it progressive. There were no intestinal ulcerations, no evidences of tuberculosis within the gut.

The iliac artery and vein on the side of the gangrenous leg were both completely thrombosed. The degree of arteriosclerosis at this point was very slight. A portion of the thrombus in the iliac vein had broken loose, and the final event, not suggested at all in the history, was a huge pulmonary embolus completely blocking both branches of the pulmonary artery.

I think there is no doubt that the gangrene of the right leg was due to the thrombosis of the iliac artery, but why that occurred I have no idea. There was no evidence of involvement of the wall of the artery by any tuberculous glands, and the degree of arteriosclerosis seemed quite insufficient to explain it.

Dr. Cabot: It seems to me a very interesting case. In acute cases we often find blocked arteries causing gangrene, the block due in the first place to arteriosclerosis with or without diabetes, and secondly to embolism. This clearly was not either, and there is no third that is recognized clinically in acute cases. It seems to me very important that we should remember this case as stretching our ideas about peripheral gangrene. Of course Buerger's disease and syphilis with syphilitic changes in the arteries are the other things that come into our minds, but those can be ruled out. There is a great deal to say about peripheral thromboses. Sometimes they are multiple and wholly venous, and sometimes do not lead to gangrene or infection and finally clear up.

DIAGNOSTIC FORUM

DERMATITIS AND DROWSINESS

Case No. 15031, Case Records of Massachusetts General Hospital, from New England Journal of Medicine, January 17, 1929).

CASE HISTORY

An unmarried English nursemaid thirty years old entered May 24 complaining of inflammation of the skin of one month's duration. She was very drowsy. The history was obtained in part from her sister, in part from the dermatologist who sent her in.

Six months before admission she began to have frequent head colds, to menstruate every two weeks, and to have pimples during catamenia. Since the onset she had been losing weight. These symptoms continued four months before she consulted a physician. Under his treatment she felt better for a while, and the skin condition improved. After two weeks, however, she felt weak, tired, and had no appetite. Her skin at that time was clear. Four weeks before admission she had generalized itching, weakness and loss of appetite and sleep, and an eruption of dark red pin-head sized spots on the arms, neck and face. Her temperature was 102°. A diagnosis of measles was made, though she had no cough or coryza. She was in bed for a week with fever, which gradually quieted down. She was up and about for a while. A week before admission she began to have considerable oozing about the neck and axillae. This persisted until May 12. A later history showed that on April 6, thinking she was pregnant, she had a curettage performed. From this time she felt very badly. She took medicine by mouth, but had no injections. Two weeks before admission she felt much worse, and entered another hospital. On admission there she presented a picture of a generalized exfoliating skin condition with marked crusting, oozing and swelling about the face, neck, arms, hands and axillae. Under treatment this gradually subsided to the dry exfoliative stage in which she entered the Massachusetts General Hospital. She had the same bronzed color which she had on admission here. She ran a temperature reaching 104° for the first week. Her elimination always remained low. She had nausea. For the past week she had had many loose movements and cramps in the abdomen. No microscopic blood or pus was found in the stools. She became more stuperous and passed smaller and smaller amounts of urine.

Clinical examination showed an emaciated and very drowsy woman lying in bed. The breath had a sweetish odor. The skin was dry and brownish throughout, and showed extensive exfoliation. The sclerae and conjunctivae were injected. The base of the right lung posteriorly showed diminished resonance, breath sounds and voice sounds. The heart showed no enlargement to percussion. The apex impulse was not seen or felt. The action was rapid. The sounds were not of good quality. The blood pressure was 95/65. The abdomen was distended and tympanitic. The liver edge could not be felt because of tenderness and a suggestion of spasm in the right upper quadrant. The upper edge of liver dullness was in the 7th space. Both labiae were swollen. All the extremities showed coarse tremor due apparently to weakness. The pupils were normal.

Urine: amount 5 to 35 ounces, specific gravity 1.012 to 1.013 at all of 6 examinations, cloudy and alkaline at all, a trace of albumin at all, red cells and leukocytes at three, loaded with both at one. Arsenic determination test on the urine showed no arsenic. Blood: 11,500 leukocytes. polymorphonuclears, 76 per cent, hemoglobin 60 per cent, reds 3,270,000. May 28 non-protein nitrogen 300, blood sugar 196 milligrams, blood chlorides 605 milligrams; May 31 non-protein nitrogen 230, blood sugar 117, creatinin 12.0 milligrams. Smear from throat negative. Stools negative.

Temperature 98° to 100.3° for the first two days; then 96.5° to 99°, with a terminal rise to 103.5°. Pulse 90 to 130. Respirations normal except for a terminal rise to 40.

After a subpectoral of 2000 cubic centimeters of glucose in saline the patient felt and appeared better. The pulse was stronger. She voided only once in the first two days. She was catheterized and an ounce of clear urine withdrawn. May 26 she was given a second subpectoral. She felt much better, was less drowsy, passed more urine and had less diarrhea. The blood pressure was 110/70. There was cavernous breathing above the level of dullness at the right base. A medical consultant suspected pathology at the right base and asked for x-ray examination. He found an apical systolic murmur and a systolic with a high pitched diastolic at the base, but found no evidence of pericardial effusion. X-ray examination showed some decreased radiability of the right base The diaphragm however was visible The costophrenic angle was clear

June 4 the patient went into coma, was incontinent, and showed considerable twitching. A subpectoral of 1600 cubic cenitmeters was given with no subjective improvement. The coma deeped, the twitching increased, the temperature, pulse and repirations rose. June 6 she died.

The following discussions of this case were given by Group 1 before the Yavapai County Medical Society and Medical Officers of Fort Whipple, at Prescott, Ariz., at tehir meeting of February 11, 1930:

DR. C. E. YOUNT

We have found this to be a particularly interesting case and one the like of which we have not seen in our discussions before.

The chief symptom, the cause of admission, is a chronic skin affection of one month's duration. Six months is the time covered by the history. Death occurs one month after the acute dermatitis, which has its onset with fever.

In considering skin disease, we naturally ask ourselves whether the case is of extraneous origin or whether it is something within. We are given a dermogram or demograph in this wonderful organ, the skin, but at present I think none but the most skillful dermatologists can read all of them, and for use in general work some elucidation of these dermograms should be had. In analyzing a condition of this kind, we know that in certain clinical pictures we may have very definite objective evidence of cause; may have urtcaria due to scores of foods and other substances producing urticaria; the mechanism prevailing anaphylatic, but the agents are legion. On the other hand, we may have a single agent, such as arsenic or the bromides, which will produce several types of skin diseases, as erythema, vesicles, pustules, granulomas, pigmentation, etc. "If so many factors may produce a single objective phenomenon like urticaria, and one factor like bromide or arsenic produce so varied a series of phenomena, it is easy to grasp that concepts in clinical dermatology are subject to enormous excursions into the field of error, largely subjective, but it is worse when it is objective." What we need, then, in this case are facts and fgiures to determine the relationship between skin and genral diseases. and facts and figures are precisely what we lack. For instance, toxic erythemas, arising as they do in sepsis and drug intolerance, as arsenic, indicate that our ignorance of the causation of these dermatoses depends entirely upon our inability to discover these facts.

Let us consider the two predominant types of skin diseases exhibited by our case: (1) Exudative inflammations, (erythemas with true exudation, true skin changes with inflammation), such as erythema multiforme, erythema scarlatinoides, pellagra, urticara pigmentosa, pityriasis rubra Hebra, dermatitis exfoliativa, prurigo. lichen planus, psoriasis, eczema, dermatitis medicamentosa; (2) the pigmentation group. One dermatologist tells us, "So far as we know, there is but one skin pigment, and all shades of color of all human races, as well as of all normal individuals of the same race and nearly all abnormal pigmentations depend on quantitive, not qualitative, changes in this one pigment, which is normally most abundant over those surfaces of the body usually exposed to the sun and wind, in the eye-lids, certain hairy regions,—axillae, genitalia. linea alba, etc."

Under pigmentation skin disease we have:

(1) Addison's disease. We should like to classify our case in the Addison type of disease, certainly in the hypoadrenia group. We think hypoadrenia is a factor but our case doesn't furnsih enough ashenia and is too acute to fit the usual picture of Addison's disease, although generalized bronzing of the entire body is found only in two groups,—arsenical poisoning and Addison's disease.

(2). Hemachromatosis, the bronzed diabetes of Osler; there is generalized bronzing, but this case is not in that class if the urine findings are correct.

(3). Pernicious anemias; usually lemon coloured, not bronze, but if much arsenic is used, the skin may be darker.

(4). Arsenic; dermatitis and intense pigmentation.

(5). Pellagra; district and seasonal, asthenia not marked (should be very marked in Addison's syndrome), the discolouration of the skin is strikingly symmetrical and delimited.

(6). Jaundice group; we would expect the sclerae to show jaundice and to find bile in the urine.

(7). Cachexias; tuberculosis, particularly tuberculous peritonitis. involving the chromataffin system and the intestines; carcinoma; malaria; syphilis.

(8). Eczema; scleroderma; melanosarcoma.
(9). Neoplasms; especially benign forms; melanotic case; lymphoma; benign tumors of tubes and ovaries; psuedoleukemia.
(10). Graves disease; spots and patches.
(11). Pregnancies; chloasma patches.
(12). Gastro-intestinal disease; ulcer and dilatation.
(13). Cardiovascular; arteriosclerosis; chronic heart disease; nephritis.

Within these two groups of broad classification of skin disease we would try to fit our own case.

This young woman, six months previous to her death, noticed frequency of menstruation, frequent head colds, and began to develop acne. She continued with these symptoms, not doing much about them until after four months when she consulted a physician. She received treatment and seemed to improve; skin cleared up. Then, thinking she was pregnant, about April 6th she had a curettment and went down hill rather rapidly after that. Two weeks later she developed what seemed to be measles; there was temperature of 104, gradually subsiding in one week. Measles and scarlet fever have been known to intensify the symptoms of pellagra and, in view of the subsequent history of the skin disease, I cannot find any particular skin condition that will fit except pellagra, and there is nothing said about delimited or symmetrical outline over the body, so we will have to rule out pellagra.

Two weeks later there is the appearance of skin condition very different from the original one, also very different from the outbreak of "measles." At this time there is much in the case to refer it to either pellagra or Addison's disease, but it could go out of that bronzed class and fall easily into one of the others of bacterial origin, like tuberculosis or septicema.

The fever ranges rather high at that time, 104 is the highest, and she developed the skin condition rater rapidly. There is moisture, exudative dermatitis, with moisture around the neck, face, axillae and arms. She was admitted to hospital with generalized bronzing. If that means what it says, it certainly points to Addison's disease, yet she is not asthenic and the condition is acute, not chronic as commonly found in Addison's. Pellagra is ruled out except the typhoid type when you may have almost anything and death is quick as in this case; but we can not fit pellagra into this case. She was given treatment with some arrest and drying of the skin but still had, when admitted to the Massachusetts General Hospital, a decided exfoliative dermatitis of some kind. Call it eczema if you will.—it is exfoliating, it is desquamating, it is true dermatitis with a generalized bronzing, our patient growing rapidly worse, with death in twelve days after admission to the Massachusetts General Hospital.

Dr. Allee will give the positive findings based on the facts and figures available, and our group diagnoses.

DR. GAIL D. ALLEE

You will remember, in reading this history, that there is nothing concerning the past history of the case. The information recorded was obtained from the family doctor and from her sister. Evidently there was nothing very striikng or her sister would probably have known about it.

This case starts in with excessive menstruation, every two weeks, and a history of pimples during menstrual period. The next episode occurs seven weeks before admission, when she has a curettage because she thinks she is pregnant. About two weeks later she has a skin eruption, which is diagnosed as measles. Two weeks later she has a terrific dermatitis of exfoliative type. In the physical examination, the interne describes perfectly some fluid in the right base, diminished breath sounds, diminished voice and resonance. The consultant upsets a diagnosis of fluid by calling for an x-ray, which reports that the costo-phrenic angle is clear. The shadow in the lower right lobe is evidently localized pathology in the lung. Could this have been tuberculosis that started off so quietly and quickly became so serious that the patient died? It seems impossible; there is no evidence of fever until along towards the last, no evidence of cough, expectoration, night sweats and other symptoms of tuberculosis. It certainly does not sound very probable that it could have been pulmonary tuberculosis.

The distended abdomen, tenderness and other abdominal symptoms directs our attention to the abdomen as a possible site of tuberculosis, but evidence of peritoneal involvement is not at all strong. If we could make that diagnosis it would help us with that bronzing of the skin, which is a very striking symptom in this case. Anything that irritates the chromataffin system may cause bronzing in the skin. That is where a diagnosis of tuberculous peritonitis would come in handy as explanation. It could not be Addison's disease; it is too acute and there is too much evidence of infection.

You will recall also that the liver dullness extended down to the seventh interspace, which is rather low, especially if she were a modern lady and did not lace tightly. This apparently small liver makes us wonder if this bronzing could have been deep icterus, accompanying an acute yellow atrophy of the liver. She had a curettage and possibly chloroform was used. She became decidedly worse about a week or ten days after the curettage. Chloroform is one of the etiological factors in yellow atrophy. The chief objection to such a diagnosis is that with acute yellow atrophy there is a generalized icterus; but ni this case we are told that the sclerae are not yellow, they are spoken of as injected, which I interpret to mean more or less redness from enlarged blood vessels.

Let us dispose here of pernicious anemia. The blood picutre indicates quite clearly a secondary and not pernicious anemia. We want to dismiss pernicious anaemia and if that is the diagnosis we will confess we are away off.

It is rather hard to give up arsenic poisoning until we reflect that arsenic would persist in the urine for months after the system is saturated with it.

In the laboratory reports everything indicates that we have a pair of kidneys that are not doing anything; they are clear out of commission; nonprotein nitrogen 300, creatinin 12, which is out of reason if we are getting any service out of the kidneys; also there is blood in the urine. Whatever else she may or may not have, it is clear that she does have a nephrieis.

We have a diagnosis of dermatitis exfoliativa already given and we think the history indicates that this woman's skin is a mirror for everything that happens to her. The excessive menstruation is probably due to an endometritis and on top of that, she has a curettage for suspected pregnancy. In all likelihood there was more or less infection or the absorption of toxins which stimulated the skin still further. Then the kidneys started with this nephritis, which grows worse and is reflected in the skin. Possibly that bronzing which occurs is due to some drug applied as external treatment. The nephritis is enough to account for all the abdominal symptoms, diarrhea, distention and cramps. The nephri-

tis can also account for an endocarditis, which we rather believe that she has. The consultant found some abnormal diastolic heart sounds. Outside of that there is very little evidence of endocarditis except that very low blood pressure, which may mean an accompanying myocarditis. Evidently there is something disturbing the heart muscle very seriously and we rather believe it is endocarditis. The findings in the lung we interpret as broncho-pneumonia. If our theory of endocarditis is correct, and we think it is possible, there might have been a lung abscess from embolus, or might possibly have been an infarct. It is not at all impossible that in the last few days of her life she may have been generally septic, and we will not be surprised if she shows a little peritonitis.

Our diagnosis is: Nephritis, glomerular
Broncho-pneumonia
Endocarditis
Dermatitis exfoliativa
Sepsis (doubtful).

The following discussions of the case were given before the Clinical Club of Phoenix, at their regular weekly Monday noon meeting:

DR. S. I. BLOOMHARDT

Concerning the dermatitis, I am more or less at a loss as my knowledge of skin lesions is even more unfortunate than my knowledge on some other medical and surgical subjects. Six weeks before admission, she had a curettment, thinking she was pregnant, and from this time on she felt very badly. Four weeks before admission, two weeks later in fact, she had a generalized itching, weakness and loss of appetite and sleep, and an eruption on the arms, neck and face, a temperature of 102° continuing for one week. A diagnosis of measles was made, but could this not have been a toxic erythema with the temperature continuing for a week, with itching, etc.? Two weeks before admission, she entered another hospital presenting a picture of generalized exfoliating skin condition with marked crusting, oozing and swelling about the face, neck, hands and axillae. This subsided under treatment and presents very much the picture of exfoliative dermatitis. The nausea and diarrhea could be associated with her extreme toxemia. There are a large group of toxic erythematous eruptions; in all these conditions, there is some evidence to support the view that a hemolytic streptococcus may play a part in the causation. The organism has actually been recovered and grown in pure culture from some of the lesions. In a large percentage of acute nephritis cases there is a history of tonsillar trouble, dental sepsis or other infectious foes; also, the causative toxin may be medicinal or metabolic. Certainly, the most frequent causative agent in acute or subacute nephritis, especially the acute glomerular nephritis has something to do with the various strains of streptococcus. Just in what manner this organism produces the lesion we do not know as no evidence has been obtained to show that the streptococcus causes the nephritis by actual invasion of the kidney, for blood cultures and urine cultures were negative. Some John Hopkins workers put forth the theory that the "streptococci produce so-called filtrates of considerable potency and that it seems possible that such toxins liberated by the growth of streptoccoci and eliminated through the kidney might cause glomerular nephritis in patients rendered highly susceptible in some way to these toxins." The throat culture was negative, she had had a curettement and from this time on, she felt badly and ran a temperature. We have mention of both labia being swollen, but no mention of edema elsewhere. There is a possibility of her having an infected uterus, streptococcic, although if such were the case, I believe more mention, with all the studies on the patient, would be made of it.

A patient with extreme drowsiness, breath has a sweetish odor, heart which is rapid, muscle sounds of not good quality, possibly extremely toxic, a blood pressure of 95/65 (this rather does not seem so much in harmony with the rest of the picture, unless B. P. taken at a time of extreme toxic state of heart and laxity of blood vessels), urine ranging in amount from 5 to 35 ounces, a specific gravity of 1.012 to 1.013 at all of six examinations, no trace of albumin, red cells and leucocytes at three, loaded with both at one. No arsenic in urine. A marked anemia, non-protein nitrogen from 300 to 230 mg. Blood sugar 196 and 117 reg. Blood chlorides 605 mgs. Creatinin 12 mgs. Temperature 98 to 100-103 rather a picture of acute nephritis with very severe impairment of renal function, extreme retention and uremic poisoning when the urine becomes approximately of the same molecular concentration as the blood, whose specific gravity is about 1.010, and a figure very close to this is the highest that can be attained by a patient with severe impairment of renal function. As water excretion becomes more and more impaired the minimum also approaches this figure and the patient, as in this case, may go on with a specific gravity between 1.008 and 1.012. This patient evidently could no longer raise the urinary concentration nor could she regularly raise the volume. A nonprotein nitrogen of 300 mg. on May 28th or 230 three days later is excesisvely high, as is a creatinin of 12 excessively high. It is my understanding that cases of definite renal involvement showing a nonprotein nitrogen of 150 mg. or more per 100 cc. of blood or a creatinin value of over 5 mg. per 100 cc. of blood should always put us on our guard as to the prognostic seriousness of cases showing such figures. The laboratory tests were made after two subpectoral injections of glucose in saline were given after which the patient felt much better, was less drowsy, passed more urine and had less diarrhea. A dullness at the right base, x-ray showing some decreased radiability, would mean a terminal pneumonic process. In many of these cases of uremia, the buccal mucous membrane is red, the tongue is enlarged and covered with a grayish, sticky coating; there is nothing of this mentioned in the physical findings. There is no mention of a blood diazo test which I believe is a valuable aid in the diagnosis and prognosis of renal insufficiency. Blotner & Firtz tell us "it is a test of practical value, being a quick step toward a differential diagnosis between uremic coma and coma of other than renal origin; it is a test of clinical significance as its presence is always an ominous sign, suggesting a short span of life for the patient."

Evidently, arsenic poisoning from some source was thought of as we have a laboratory report to the effect that arsenic determination test on the urine showed no arsenic. It is an etiological factor in nephritis and in the genesis of certain eczemas, but I believe when the cause, it can be demonstrated in the urine.

Diagnosis:
Uremic poisoning;
Acute nephritis;
Terminal basal pneumonia;
Toxic erythema, exfoliative dermatitis.

DR. W. WARNER WATKINS

According to the history of this patient, the chief symptoms in the order of their appearance were head colds, menorrhagia, pimples, loss of weight, loss of strength. pruritus, insomnia, loss of appetite, fever, dermatitis, diminishing urine, gastro-intesinal upset, drowsiness.

The loss of weight, strength and appetite, the fever, the dermatitis with itching, the diminishing urine and the drowsiness would seem to be the suggestve symptoms. If we are going to attempt to explain all the symptoms on the basis of a single diagnosis which we should at least attempt, the dermatitis seems to be the best symptom to analyze.

The itching varieties of dermatitis practically rule out some very common varieties, but we should not be too sure of this.

Any dermatitis requires that syphilis be ruled out; though this woman was unmarried, yet she had sexual exposure, because she feared pregnancy and was curetted. Aside from this, we have little to suggest syphilis.

The only other non-itching dermatitis which occurs to me as possible would be pellagra. While the distribution of the lesions would be atypical for pellagra, we do have such cases. She has the insomnia, drowsiness, and gastro-intestinal upsets which we expect in pellagra, but without going into all the reasons for the decision, I have rejected pellagra as a diagnostic possibility, with an uncomfortable feeling that I may be wrong.

Of the itching varieties of dermatitis, the parasitic, dermatosic (eczema, mycosis fungoides, lichen, etc.), cholemic, phramaceutic and neuropathic can all be fairly well ruled out, with a reservation about mycosis fungoides and chronic arsenic poisoning. That leaves us the allergic type, the azotemic, the diabetic, and any unknown. On the basis of the history, we could have either of these.

The cinical examination brings us the symptoms of sweet breath, some lung symptoms, feeble heart action, low blood pressure, distended abdomen tremor.

The clinical examination does not rule out either of the three possibilities mentioned, but adds another. The low blood pressure, feeble heart action without evidence of heart disease, the tremor of extremities bring into the picture Addison's disease.

The laboratory findings add the information that there is a small output of low specific gravity urine, with trace of albumin, no mention of glycosuria, a secondary anemia, very marked retention of nitrogen products in the blood, with slight excess of sugar in the blood.

Without discussion of the reasons therefor, will at this time drop the allergic type of dermatitis from consideration; it would take a long time to tell just why, but there are reasons good enough for me.

I should say that we would need now to differentiate between three possibilities,—diabetes, nephritis with uremia, and Addison's disease.

For diabetes, the sweetish odor of breath, the loss of strength, the fever, the drowsiness and terminal coma, slight excess of sugar in the blood, the aggravated type of dermatitis with bronzing, the low B. P,. the improvement after glucose are all in accord. Against diabetes will be the absence of polyuria, the low sp. gr. of urine, the apparent absence of glycosuria, and the relatively low blood sugar in a patient shortly to die in coma. All told, I vote against diabetes.

Azotemia from nephritis is supported by gastro-intestinal symptoms with diminishing urine, the edema, the urinary findings of small output through low specific gravity, trace of albumin, very high nitrogen retention, remaining 230 mgm. after subpectoral injections. There is certainly an azotemia whether or not this is cause of the symptoms or death. The drowsiness with coma could be explained by a terminal uremia. Against nephritis is the fever, the low blood pressure, the weak heart action, and the very aggravated type of dermatitis, with bronzing.

For Addison's disease, we have the necessary classical symptoms of asthma, anemia, feeble heart action, low B.P., intestinal disturbance, bronzing of skin, etc. In fact, there is hardly any symptom presented which Addison's disease will not explain, except the extreme dermatitis. The intermittent character of the disease, also is in accord with Addison's as are the terminal symptoms. The symptoms which are not in accord are the laboratory findings, the very high nitrogen,—although there is an increase in Addison's. There is said to be a hypoglycemia in Addison's and this patient has a slgiht excess of sugar.

Having reached the end of my allowance of time for this case, without further, discoursive straddling, will place my bet on Addison's disease, with an azotemia from accompanying nephritis. The lung findings will lay to hypostasis from the feeble circulation.

Diagnostic conclusions on this case by members of the Club were as follows:

Dr. H. Randolph—Uremia coma; chronic nephritis.

Dr. V. Randolph—Addison's disease.

Dr. F. C. Jordan—Nephritis.

Dr. T. T. Clohessey—Visceral and skin tuberculosis.

Dr. J. M. Greer—Diabetes; nephritis; dermatitis exfoliativa.

Dr. O. H. Brown — Nephritis, subacute; subphrenic abscess; allergic dermatitis.

Dr. A. C. Kingsley—Nephritis.

Dr. Fred Holmes—Addison's disease; diabetes.

Dr. R. J. Stroud— Nephritis; dermatitis exfoliativa.

Dr. F. J. Milloy—Chronic amebiasis; abscess of liver.

The following discussions of the case were given at the Clinico-Pathological Conference at the Massachusetts General Hospital:

DR. RICHARD C. CABOT

I do not see how we can tell about the measles. It does not sound like it in the absence of any catarhal symptoms.

The outstanding facts are the skin condition, which sounds like an exfoliative dermatitis of the type we often get after a dose of salvarsan or other poisons, plus an increasing drowsiness, with deficient excretions. I have not paid any special attention to the brown color of the skin because that is so common in cases of this dermatitis. The blood pressure was notably low, which with the brown color, which I have just said is of no importance, might make us think of Addison's disease. We must watch for other evidence of this as we go on.

They are looking carefully as to the size of the liver in relation to the possibility of an acute hepa-

titis, which often goes with exfoliative dermatitis as a result of an overdose of salvarsan, but ordinarily accompanies a jaundice which has not as yet been mentioned and I suppose was not there.

Of course we cannot tell much about the statements as to the urine because we do not know whether these are catheter specimens. The apparent fixation of gravity is important, although fixation at that point is not so important as at a lower point.

Arsenic was not being excreted at this time, which only shows probably that it had not been given very recently.

A non-protein nitrogen of 300 certainly ought to mean something. That is not healthy. The creatinin ought to be from one to two. It is greatly increased, as is the non-protein nitrogen.

She wa shere two weeks. She had a good deal of normal temperature. I doubt whether the breathing was cavernous. That is just a question of fact. It does not seem likely there was a cavity there, and we do not get the cavernous breathing under other conditions.

From the x-ray report there probably was no fluid in the plurae.

DIFFERENTIAL DIAGNOSIS

This is the picture which for so many generations has been known as uremia. We do not know today just what we mean by uremia from the pathological point of view, but we still continue to use the term for this clinical syndrome with the implication of a nephritis as its cause. It seems to me we have no reason to doubt the diagnosis of a severe nephritis, though we have a low blood pressure. The only thing in my mind is the lesion we had a week or two ago, nephrosis or tubular nephritis, which can perfectly well come in a case presenting the facts that are given here. Nephrosis does not usually raise the non-protein nitrogen, and the fact that very is so very high makes me think tha tthe glomeruli are affected,—that it is not merely a tubular process.

I suspect then, that the patient had taken some poison, perhaps arsenic in the form of salvarsan, that led to an exfoliative dermatitis and nephritis. I do not think we have any good evidence of hepatitis, although that is possible.

What is this lesion at the base of the lung? I do not know, unless it is a pneumonia. I do not know just how that plate corresponds in time with the temperature.

So it seems to me that some obstructive type of kidney lesion ought to be found, and I believe it will not be nephrosis but nephritis. That is, it will not be confined to the tubules. We have no evidence of a terminal pneumonia on the right. That is all that I can say.

A Student: Acute endocarditis?

Dr. Cabot: I am glad you brought that up. There is a high-pitched diastolic murmur at the base. That ought to be due to a definite valvular lesion, acute or chronc. We do have functional diastolic murmurs, but ordinarily with very much more anemia than there is here. They occur chiefly in intense anemia. This is not intense, only moderate, and I do not believe it is a functional murmur. The question is whether this report is a mistake. But if the murmur was really there I think it is a case of acute or chronic lesion on the aortic valve. It looks more like an acute lesion.

A Student: Would you consider tuberculosis,—tuberculosis of the kidney and adrenals, thereby producing Addison's dsiease?

Dr. Cabot: I have never seen that combination. I do not think tuberculosis of the kidney ever gives this urinary picture. But I think we ought to consider Addison's disease a little further. We diagnose Addison's disease on the basis of changes in the skin, low blood pressure, and certain gastric and cardiac symptoms, especially fainting and vomiting. Pigmentation inside the mouth is the most reliable physical sign. Here we have nothing except pigmentation and one rather low blood pressure, the other being within normal limits. And we have good reason to explain the pigmentation of the skin from the other skin lesion which she has. So I should say I do not think we have to consider tuberculosis of the adrenal here.

INTERPRETATION OF X-RAY

The lung findings suggest consolidation rather than fluid.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Dermatitis exfoliativa.
Uremia.

DR. RICHARD C. CABOT'S DIAGNOSIS

Nephritis, probably due to arsenic.
Dermatitis exfoliativa.
Possibly terminal pneumonia.
Possibly acute or chronic endocarditis of the aortic valve.

DR. T. B. MALLORY

ANATOMIC DIAGNOSIS

1. Primary disease.
   Interstitial nephritis.
2. Secondary or terminal lesions.
   Interstitial pancreatitis.
   Dermatitis exfoliativa.
   Infarcts of the spleen.
   Bronchopneumonia.
3. Historical landmarks.
   Chronic inflammation of the duct of Wirsung.

This case was an even rarer type of renal lesion than the nephrosis which was seen recently at the Brigham Hospital. It was a generalized interstitial nephritis, a disease that unquestionably was very much commoner twenty years ago than it is now. The postmortem records at the City Hospital show from 1900 to 1910 a number of cases every year of interstitial nephritis, almost invariably in cases convalescing from scarlet fever. I believe I am correct in saying that in the last fifteen years there has not been a case at the City Hospital. I am sure that in ten years there have been no cases here. The condition occasionally does occur in cases in which scarlet fever cannot be proved. It is however rare, and I think that the suggestion that the original rash may have been scarlatinal is given extra strength at any rate by the character of the nephritis here, although we are not able to prove it.

The kidneys were very large, the pair weighing together 600 grams. The capsules strpped very readily, leaving a smooth, somewhat grayish-red surface with an appearance suggesting a diffuse infiltration, very much the type that is seen in a leukemic infiltration of the kidneys. Microscopic examination showed negative glomeruli, for the most part negative tubules, and an extremely diffuse infiltration of lymphocytes, plasma-cells and a few eosinophiles. Although the tubules did not show much it is not fair to say they were entirely negative. In some places there are leukocytes present in the lumina, and one also finds rare mitotic figures in the epithelial cells, signifying regeneration and presumably previous injury to the tubular cells. The exact mechanism of the production of an interstitial nephritis is still undecided. Some authorities

claim it starts about the blood vessels, others that it represents a healing stage, perhaps of a tubular nephritis. Some of the findings here were suggestive of that point of view. I think we can definitely rule out a healing bichloride of mercury poisoning on account of the large amount of calcification that develops in such cases.

The heart was very small, weighing only 210 grams, the valves entirely negative.

There was a bronchopneumonia at the base of the lungs. The liver was large and showed a very marked degree of fatty infiltration rather similar to what is often seen in cases of nephrosis, though by no means characteristic of that condition.

There were two small infarcts in the spleen. The pancreas, surprisingly enough, showed also a very marked interstitial pancreatitis.

No further chemical examinations were made on the case, as I had been assured that arsenic and mercury had been ruled out.

Dr. Cabot: As you put all the facts together here, what do you think was the relation of this skin condition to the kidney condition?

Dr. Mallory; I think it was really very suggestive that she may have had a scarlet fever. I am unable to explain the exfoliative dermatitis. That is of course very suggestive of arsenic, but unquestionably can occur without it.

Dr. Cabot: This type of nephritis is not the type Dr. Councilman described twenty years ago as "acute interstitial nephritis?" That was in connection with diphtheria as well as scarlet, wasn't it?

Dr. Mallory: The majority of cases follow scarlet fever, though cases do occur in diphtheria, erysipelas or any acute disease such as measles in which septic, particularly streptococcic complications develop. The condition has not been reported following any nonbacterial toxemia to my knowledge.

Dr. Cabot: I still feel that the skin lesions here described are extremely unlike scarlet fever, though I have no proof that she did not have it. Of course Dr. Mallory is on perfectly safe ground when he says people can have scarlet fever without a rash. That has been proved in experimental scarlet fever. Therefore the absence of rash is not against scarlet fever.

MINUTES OF THE MEETING OF THE THIRTY-NINTH ANNUAL SESSION OF THE ARIZONA STATE MEDICAL ASSOCIATION, HELD IN PHOENIX, APRIL 24 TO 26, 1930.

MEETING OF THE COUNCIL

Meeting of the Council was called by the president, Dr. S. H. Watson, to meet at 8 p. m., April 23rd, at the Hotel Westward Ho. Council members present were: Dr. S. H. Watson (president), Dr. J. M. Greer (president-elect), Dr. D. F. Harbridge (secretary), Dr. C. E. Yount (treasurer), Drs. W. C. Todt, C. A. Thomas and W. W. Watkins (councilors).

Minutes of the previous Council meeting were read and approved.

Dr. W. C. Todt, councilor for the Northern District, reported that he had held a council district meeting in Winslow during the year.

Dr. C. A. Thomas, councilor for the Southern District, reported that, so far as he knew, only Pima County was holding regular meetings; that he had some matters regarding Santa Cruz County to report which he would hold until new business was in order.

Dr. W. W. Watkins, councilor for the central district, reported that only Maricopa County was holding regular meetings in his district.

Dr. Watkins was asked for a report on the official journal—Southwestern Medicine. He made a verbal report, with the explanation that he was not the representative of this Association on the journal staff, that place being held by Dr. O. H. Brown as associate editor and by Drs. D. F. Harbridge and C. E. Yount, as members of the Board of Managers. He commended the work of Dr. Brown very highly. Dr. Todt suggested that the names of the various county societies, with their members, be published in the journal at least once a year, for reference.

Dr. C. E. Yount made recommendation that the chairmen of the Committees on Public Welfare and Medical Defense be made regular members of the council, and that amendments to the Constitution providing for this be offered.

The report of the Treasurer, Dr. C. E. Yount, was presented by him, with explanations of method of handling funds; this report had been audited by the Auditing Committee appointed by the president. The report is appended to these minutes.

Report of the Secretary, Dr. D. F. Harbridge, was read and approved (see below.)

Report of the Committee on Medical Defense was presented for the information of the Council (see below).

Under the head of New Business, the application of a group of doctors in Santa Cruz County for a charter as a constituent county society of the Association, was presented. Dr. Harbridge, Dr. Thomas and Dr. Watson stated that they had visited the county, that the old society was entirely inactive, and recommended revocation of its charter, and the granting of a new charter. After considerable discussion on constitutional points, it was moved and seconded that the new organization in Santa Cruz County be accepted as the constituent organization from that county. This motion was carried. It was then moved and seconded that the Council accept and approve the bylaws of the new Santa Cruz Medical Society, as presented. This motion carried.

Council adjourned.

HOUSE OF DELEGATES

First meeting, Thursday, April 24.

Called to order by the president. Members of the Council present were Drs. S. H. Watson, J. M. Greer, D. F. Harbridge, C. E. Yount, C A. Thomas, W. C. Todt, W. W. Watkins. Delegates present as follows: Coconino County, Dr. F. P. Manning; Graham County, Dr. R. C. Dryden; Cochise County, Drs. R. B. Durfee and Robt. Ferguson; Gila County, Drs. C. I. Irvin and C. Gunter; Maricopa County, Drs. Win Wylie, H. T. Bailey, H. B. Gudgel, J. J. McLoone, R. T Franklin, G M Brockway; Yuma County, Dr. H. A. Reese.

Minutes of previous meeting were read and approved.

President appointed as the Committee on Necrology, Drs Todt. Looney and Manning.

Committee on Public Welfare reported by Dr. Clarence Gunter, the chairman. The matter of the Basic Science Law had been considered by them, but they were as yet not satisfied about the desirability of this legislation in Arizona. It was moved and seconded that the matter be left with the committee and that they continue their work until next January. Motion carried.

Committee on Medical Defense reported through Dr. D. F. Harbridge, who read the chairman's report, and the communication from the Association's attorneys, which are appended hereto. This report was approved by motion.

Dr. Watkins presented a proposed amendment to the Constitution, Art. VI, to make it read as follows: "The Council shall be the Board of Trustees of this Association and shall be the Finance Committee of the House of Delegates. It shall consist of the councilors, the president, the president-elect, th retiring president, the secretary, the treasurer, and the chairman of the Committee on Medical Defense. Five of its members shall constitute a quorum."

He also presented an amendment to Chap. VII, Sec. 3 of the By-laws by the addition of the phrase, "the president of the Association to be its chairman."

Also an amendment to Chap. IX, Sec. 3 of the By-laws, by inserting the phrase "licensed to practice medicine in Arizona," so that it shall read, "Each county society shall judge of the qualifications of its members, subject to review and final decision of the Council of the State Association. Every reputable physician, legally licensed to practice medicine in Arizona, who does not practice nor profess to practice sectarian medicine and who is a bona fide resident of the same county, shall be eligible to election to membership."

Adjourned.

Second meeting of the House of Delegates, Friday, April 25, 9 a. m.

Vice-president, Dr. H. A. Reese, presiding. Following delegates were present: Dr. Dryden (Graham), Drs. Gunter and Bacon (Gila), Drs. Franklin and Stroud, (Maricopa), Members of Council, Drs. D. F. Harbridge, C. E. Yount, W. C. Todt, C A. Thomas.

Committee on National Legislation submitted the report of its representative, Dr. R. J. Stroud (appended below). It was moved and seconded that the report be accepted and published.

Dr. Stroud suggested that a committee be appointed to draft a narcotic law for Arizona. It was moved and seconded that the matter be referred to the Committee on Public Welfare, with instruction to the president to request a call meeting to receive its report. Carried.

It was moved, seconded and carried that a committee be appointed to work out the organization of a Woman's Auxiliary for the State Association.

With regard to the amendments to the By-Laws introduced the previous day (vide supra), the amendment to Chap. VII, Sec. 3, referring to the Committee on Public Welfare was not adopted. The amendment to Chap. IX, Sec. 3, referring to the qualifications for members of county societies was adopted.

Dr. Yount introduced an amendment to Art. VI of the Constitution, adding to the end of that article the clause "the chairmen of the Committees on Public Welfare and Medical Defense," which will make these chairmen members of the Council.

Dr. Stroud brought up the matter of a medical booth at the State Fair, and it was moved, seconded and carried that the State Health Officer have the approval of the House of Delegates in this matter.

Adjournment.

Open Meeting of the House of Delegates, Saturday, April 26, 9 a. m.

President, Dr. J. M. Greer, presiding.

Mr. George Mauk, of the Legislative Board of the Arizona Peace Officers' Association, spoke on the need of a law governing the sale of narcotics in Arizona.

It was moved and seconded that the Arizona State Medical Association extend a vote of thanks to the Medical Defense Committee for their splendid work, and also to Drs. Win Wylie and W. O. Sweek for their work in special cases. Carried.

It was moved and seconded that the Association adopt a suitable resolution expressing the sentiments of the Association toward Dr. Whitmore and his family, and their appreciation of his great services to medicine and the public good. Motion was carried, and the president appointed Drs. Wylie, Bacon and Watkins as this committee.

The Committee on Necrology submitted its report (see below), which was adopted, and the president requested the Association to stand a moment in silence out of respect for the members who have passed away during the year.

The president announced the appointment of Dr. R. J. Stroud as delegate to the American Medical Association, with Dr. Mayo Robb as alternate.

The election of officers resulted in the selection of the following:

Dr. Harry A. Reese of Yuma—President-Elect.
Dr. W. C. Todt, Kingman,—Vice-President.
Dr. D. F. Harbridge, Phoenix—Secretary.
Dr. C. E. Yount, Prescott—Treasurer.
Dr. C. A. Thomas, Tucson—Councilor of the Southern District.
Dr. F. P. Manning, Flagstaff, Councilor of the Northern District (to succeed Dr. W. C. Todt, resigned.)
Dr. R. J. Stroud, Tempe—National Legislative Committeeman.

Drs. Smelker and Gustetter extended the invitation of Nogales for the 1931 meeting. Upon motion, this invitation was accepted.

A vote of thanks was extended to the Maricopa County Medical Society for their fine entertainment of the 1930 meeting.

Adjournment sine die.

SCIENTIFIC SESSIONS

Scientific Session was opened on Thursday forenoon, at ten o'clock, with president, Dr. Samuel H. Watson of Tucson, in the chair. Invocation was given by Rev. Fr. Vaughn of Brophy College. Address of Welcome was given by State Senator Celora M. Stoddard, of Phoenix, representing Governor Phillips. The President-elect was then introduced —Dr. Joseph Madison Greer of Phoenix, who delivered his address on "Our Duty as Doctors to our Patients." (This address was published in the May issue of this journal.)

The next paper was presented by Dr. C. E. Yount of Prescott, on "Detailed Study of Tissue from Abortions." Specimens of moles and many lantern slides were shown. Discussion was opened by Dr. F. B. Sharp of Phoenix, with participation by Drs. Thigpen (Jerome), Reese (Yuma), Bacon (Miami), being closed by Dr. Yount.

There followed then the symposium on Industrial Surgery, which continued into the afternoon, as follows:

"Arthritis and Its Relation to Industrial Medicine," by Drs. John E. Bacon and Wm. B. Watts of Miami.

"Pheumoconiosis," by Drs. John W. Flinn and Robert S. Flinn, of Prescott.

"Back Injuries," by Dr. A. C. Kingsley, Phoenix.

"Infections of the Hand," by Dr. Charles N. Ploussard of Phoenix.

"Organic Internal Splinting of the Fractured or Detached Olecranon," by Dr. Wm. Lyle Bell of Oakland, Calif.

"The Relation of the Medical Fraternity to the State Industrial Commission," by Mr. John Taheney, of the Arizona State Industrial Commission, Phoenix.

The discussion of this group of papers was opened by Dr. Samuel H. Watson (Tucson), and continued by Dr. Farrington (Boulder, Colo.), Dr. W. W. Wilkinson (Phoenix), Dr. W. W. Watkins (Phoenix), Dr. C. R. Swackhamer (Superior,) Dr. R. H. Thigpen (Jerome), Dr. C A. Thomas (Tucson), Dr. C. E. Irvin (Miami), Dr E. Payne Palmer (Phoenix), Dr. John E. Bacon (Miami), and Dr. Robert S. Flinn (Prescott).

The scientific session of Friday, April 25, was given over to a Symposium on Chest Diseases, which occupied the forenoon and afternoon sessions as follows:

'Trends in Treatment of Pulmonary Tuberculosis," by Dr. Allan K. Krause, Tucson.

"Management of Artificial Pneumothorax," by Drs. Samuel H. Watson and C. S. Kibler, Tucson.

"Intestinal Tuberculosis from the Standpoint of the Roentgenologist," by Dr. John L. McKnight, Tucson.

'The Heart in Pulmonary Tuberculosis with Preliminary Report of Electrocardiographic Study in One Hundred and Four Cases," by Dr W. R. Leverton, Tucson.

The discussion of this group of papers was opened by Dr. Victor Randolph (Phoenix), Dr. Howell Randolph (Phoenix), Dr. H. L. Goss (Phoenix), continued by Dr. W. W. Wilkinson (Phoenix), Dr. S. H. Watson (Tucson), Dr. Robert Flinn (Prescott) Dr. H. A. Reese (Yuma), Dr. C. A. Thomas (Tucson), Dr. C. S. Kibler (Tucson), and Dr. J. L. McKnight (Tucson).

At the Clinical Luncheon on Friday, Dr. Ralph Homan, fraternal delegate from Texas, spoke on "Diagnosis of Tuberculosis in Childhood," (published in this issue), and Dr. Allan K. Krause continued his discussion on "Trends in Treatment of Pulmonary Tuberculosis."

The scientific session of Saturday was opened by a paper on "Pulsion Diverticulum of the Esophagus" by Dr. Charles T. Sturgeon of Los Angeles, with lantern slides. Discussion was opened by Dr. H. T. Bailey (Phoenix), continued by Dr. J. E. Bacon (Miami) and Dr. E. Payne Palmer (Phoenix).

The second paper was on "The Explanation of Symptoms" by Dr. Francis M. Pottenger of Monrovia, Calif.

At the Clinical Luncheon of Saturday, Dr. Pottenger was the speaker, on the subject of 'Visceral Pain."

At the beginnng of the afternoon session, Mr. Charles R. Holton, attorney for the Association, gave a report of last year's Medical Defense activities. The gist of his report is contained in a communication which is appended to the Report of the Medical Defense Committee (see below).

The next paper was on "Fever" by Dr. Harry A. Reese, of Yuma. Discussion was opened by Dr. Robert Flinn (Prescott), continued by Dr. Willard Smith (Phoenix) and Dr. W. W. Wilkinson (Phoenix.)

Dr. C. C. Tiffin of Seattle, who was to present the next paper on "Differential Diagnosis and Medical Treatment of Goitre" was unavoidably detained by emergency operation upon his daughter. His regrets were telegraphed to the Association. In lieu of this, Dr. S. I. Bloomhardt talked on this subject and Dr. E. Payne Palmer of Phoenix presented the "Surgical Treatment of Goitre." General discussion was participated in by Dr. Charles T. Sturgeon, (Los Angeles), Dr. Smith (Pasadena), Dr. Kimball (Yuma), Dr. Frank Milloy (Phoenix) and Dr. F. M. Pottenger.

The final paper on the program was on "Volkmann's Ischemic Contracture" by Dr. A. C. Carlson of Jerome. Report of a case with moving pictures, made by the author were shown. The discussion was opened by Dr. Edgar Brown (Phoenix), participated in by Dr. L. P. Palmer (Phoenix) and Dr. C. R. Swackhamer (Superior.)

A special moving picture film of gall bladder disease was shown.

The social entertainments included smoker for the men on Thursday evening, while the ladies were entertained at a bridge party; dinner dance Friday evening; luncheon on Friday for ladies; golf tournament for men.

REPORTS OF OFFICERS AND COMMITTEES

SECRETARY'S ANNUAL REPORT APRIL 1930.

The usual routine business of the office has been conducted. Correspondence, memberships, visit to County Society and many varied details.

One visit was made to the Santa Cruz County Medical Society to help the members to a better and more active organization. The Secretary attended the Annual Conference of State Secretaries at Chicago, Nov. 15, 1929. There was discussed at this meeting much of interest to each component State Association, the key note of which was more intensive work and a better cohesive association between the state and county organizations. The national and state organizations are no better than the conjoined efforts of the county societies make it. Proper support and enthusiasm of state journals is to be encouraged. Industrial medicine received a cordial discussion.

The lamp and screen which you ordered purchased one year ago have been secured.

Membership in good standing April 1st, 1930:

Dues received (since April 1929 meeting)....$3085.00

Expenses for which vouchers were signed....

General Expenses	1393.00
Medical defense	2216.50

Our finances are in somewhat better condition than one year ago; yet there is still room for great improvement.

It is the opinion of the secretary, based on observation of the work of the various county societies that each should exert a greater effort in the interests of organized medicine.

D. F. HARBRIDGE,
Secy. State Med. Assoc.

REPORT OF THE TREASURER

TO THE COUNCIL AND HOUSE OF DELEGATES, ARIZONA STATE MEDICAL ASSOCIATION:

I present herewith Treasurer's report for the year ending April 19, 1930. (Books closed this date.)

GENERAL STATEMENT

Total Receipts All Sources:		
Balance General Fund April 13, 1929		$ 1,891.94
Dues 1929, 12 members at $10	$120.00	
Dues 1930 238 members at $12.50	$2,975.00	3,095.00
Refund, one member bad check		12.50
Donation (Troth)		10.00
Defense Fund, Balance in Savings Bank April 13, 1929	$1,294.40	
Interest and Coupons	259.08	1,553.48
Coupons, Bonds, Dec. 1929, Deposited in General Fund		259.08
United States Bonds		10,000.00
TOTAL receipts all sources		$16,775.36
TOTAL disbursements all sources		2,968.67
TOTAL Balance all Funds		$13,806.69

ANALYSIS AND STATEMENT BY FUNDS

1. General Fund:	**Receipts**	
Balance from 1929		$ 1,891.94
12 members pro rated at $4.00		
General Fund	$ 48.00	
238 members pro rated $6.50		
General Fund	$1547.00	1,595.00
250 members pro rated at $6.00		
Defense Fund		1,500.00

Refund, one member, bad check	12.50	
Donation (Troth)	10.00	
Transfer from Defense Fund July 3, 1929	500.00	
Coupons, Bonds, deposited in General Fund Dec., 1929	212.44	$ 5,721.88
Disbursements, duly authorized—		
Paid from General Fund:		
Dr. John Alexander, traveling expenses	$ 225.00	
Louise Pearson, stenographer, Annual Mtng	125.00	
Wm. C. Todt, expenses Council meeting Flagstaff	6.50	
Martindell, Horne & Co., Treasurer's Bond	30.00	
Sloan, Holton, McKesson & Scott, Medical Defense	1,305.15	
R. J. Stroud, reimburse for telegrams	5.04	
John W. Flinn, reimburse for telegrams	2.82	
W.W.Watkins, reimburse for telegrams, etc	20.25	
Bank of Arizona, Safety Deposit Box	4.00	
Southwestern Medicine, 245 members	490.00	
Sloan, Holton, McKesson & Scott, Medical Defense	150.00	
Louise Pearson, reporting three Yavapai meetings	45.00	
D. F. Harbridge, Secretary's office expense	60.00	
Louise Pearson, reporting two Yavapai meetings	30.00	
A. C. Taylor Printing Co., Membership cards 1930	3.50	
D. F. Harbridge, reimburse for purchase record book	4.00	
Sloan, Holton, McKesson & Scott, Medical Defense	240.00	
Maricopa Medical Society, reimburse for lantern, etc.	115.00	
St. Louis Button Co., Buttons State Meeting	34.91	
Bank charge, bad check	12.50	
Louise Pearson, reporting four Yavapai meetings	60.00	
	$ 2,968.76	
April 19, 1930, Transferred to Defense Fund	1,550.12	
April 19, 1930, Balance in General Fund in in Bank of Arizona		$ 1,203.09

2. Defense Fund:

Receipts

Balance in Savings Bank April 13, 1929	$ 1,294.40	
Interest June 30, 1929	25.88	
Interest December 31, 1929	20.64	
Coupons, Bonds, deposited in Defense Fund, June, 1929	212.56	
250 members pro rated at $6.00 for Defense Fund	1,500.00	
Loan to General Fund refunded	500.00	
Due from General Fund 1929 refunded	1,239.27	
TOTAL Receipts Defense Fund		$ 4,802.75

Expenditures

Loan to General Fund July 3, 1929	500.00	
Cost of Medical Defense for current year	1,699.15	
Balance Defense Fund, Yavapai County Savings Bank April 19, 1930		$ 2,603.60

NOTE: Due Medical Defense Fund, now carried in General Fund, Coupons, Bonds, deposited in General Fund December 1929, $212.44.

CLEARING:

Due Defense Fund last year	$1,249.27	
Loan to General Fund	500.00	
Dues 250 membesr at $6.00	1,500.00	$ 3,248.27
Cost of Medical Defense current year		1,699.15
April 19, 1930, Transfered from General Fund to Defense Fund		$ 1,550.12

Itemized Statement of Medical Defense Expense:

Sloan, Holton, McKesson & Scott—	
June 20, 1929	$1,305.15
Sept. 10, 1929	150.00
Mar. 8, 1930	240.00
Rent Safety Deposit Box	4.00
	$1,699.15

Total Amount Available for Medical Defense:

Due from Gen. Fund, Coupons Dec. 1929	$ 212.44
Cash in Defense Fund	2,603.60
United States Bonds	10,000.00
	$12,816.04

Earnings of Defense Fund and Bonds Since Last Report:

Bonds, Coupons June 1929	$ 212.56
December 1929	212.44
Savings Account, Interest June 1929	25.88
December 1929	20.64
	$ 471.52

Unpaid Expense for 1930:

To Southwestern Medicine—250 members at $2.00	$ 500.00
To Secretary for office expense	120.00
Stenographer annual meeting	

Recommendations:

1. It is suggested that the Council, which is the Executive Committee of the Association, keep in close touch with the financial condition of the Association.
2. Your Treasurer recommends that the dues be continued as last year, pro rated in the same amounts—Medical Defense $6.00 and State Association $6.50.

Respectfully submitted,

C. E. YOUNT,
Treasurer.

REPORT OF COMMITTEE ON NECROLOGY

To the Arizona State Medical Association:

Your Committee on Necrology submits the following report:

Whereas, death has removed from the ranks of the medical profession of Arizona:

JAMES A. KETCHERSIDE, Yuma
EDMISTON DARRAGH, Bisbee.
ROSCOE G. BAZELL, Winslow.

And, whereas, these physicians were prominent in the activities of our State Medical Association;

And, whereas, by their kindly nature, courteous manner, and high professional attainments they won the admiration, respect and friendship of their professional confreres throughout the state;

Therefore, be it resolved, that in their death this Association has lost active and useful members, and the State of Arizona loyal and public spirited citizens;

And be it further resolved, that this resolution be made a part of the permanent record of the Arizona State Medical Association.

WILLIAM C. TODT.
R. N. LOONEY.
G. F. MANNING.

(Continued in July Issue)

FIFTEENTH ANNUAL CLINICAL SESSION OF THE AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians will hold its Fifteenth Annual Clinical Session at Baltimore, Maryland, from March 23-27, inclusive, 1931. The Lord Baltimore Hotel wil be headquarters.

Dr. Sydney R. Miller, Baltimore, as President, will have charge of the selection of the general scientific program. Dr. Maurice C. Pincoffs, of Baltimore, has been appointed by the Board of Regents as the General Chairman of the Session, and will make all local arrangements, including the making up of the program of clinics. Business details will be handled by the Executive Secretary, Mr. E. R. Loveland, from the College headquarters, 133-135 S. 36th Street, Philadelphia, Pa.

The attention of secretaries of various societies is called to the above dates, in the hope that their societies will select non-conflicting dates for their 1931 meetings.

MASONIC HOSPITAL (El Paso) STAFF OFFICERS

The following officers of the Medical and Surgical Staff of the Masonic Hospital, El Paso, Texas, have been elected to serve during the year 1930:

President—Dr. J. A. Rawlings.
Vice-President—Dr. J. H. Gambrell.
Secretary and Treasurer—Dr. T. J. McCamant.
Efficiency Committee—Drs. H. H. Varner and J. Mott Rawlings.

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Southwestern Medicine

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EDITORIAL STAFF

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DR. JAMES J. GORMAN, El Paso, Texas ..... Associate Editor
DR. ORVILLE H. BROWN, Phoenix, Arizona ..... Associate Editor
DR. L. B. COHENOUR, Albuquerque, N. M. ..... Associate Editor

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NEW MEXICO STATE MEDICAL SOCIETY

DR. C. F. BEESON ..... Roswell
DR. H. A. MILLER ..... Clovis

MEDICAL & SURGICAL ASSOCIATION OF THE SOUTHWEST

DR. E. PAYNE PALMER ..... Phoenix
DR. W. WARNER WATKINS ..... Phoenix

THE SOUTHWESTERN CLINICAL CONFERENCE

The Medical and Surgical Association of the Southwest will present another Clinical Conference, at their annual meeting in El Paso. the dates of which are to be November 6. 7 and 8.

Dr. E. Payne Palmer, the president of the Association and the Program Committee, the El Paso representative of which is Dr. Willis W. Waite, have secured a very attractive group of teachers and lecturers. The details of the program have yet to be arranged, but the speakers include such well known men as Alvarez of the Mayo Clinic, Kreuscher of Chicago. Hibbs of New York, Hermanns of St. Louis, Ochsner of New Orleans, Brenneman of Cleveland, Jackson of Denver, Roy Thomas and Sturgeon of Los Angeles. Following the idea of the president to have representatives on the program from different sections of the southwest, Dr. Leroy Peters of Albuquerque has been asked to represent New Mexico, Dr. Robert S. Flinn of Prescott and Dr. Allan K. Krause of Tucson to represent Arizona and Dr. Jim Camp of Pecos to represent western Texas.

Clinics will be presented by the El Paso County Society, assisted by the visiting clinicians and surgeons. Watch for further details in future issues of this journal.

CONVENTIONS AND CONVENTIONS

After suffering in silence for many years, we have concluded to express our opinion about one phase of medical conventions, as usually conducted in the southwest. Strange to say, it was the recounting of certain occurrences at a national medical convention, recently held, which furnished the extra weight necessary to break the figurative back,—and silence,—of this particular dry camel.

In April the Arizona State Medical Association met in Phoenix, for its annual meeting. Although the Maricopa County Medical Society has refused to authorize the expenditure of any funds from its treasury for alcoholic drinks at any meetings for which the Society is host, the committee on arrangements considered it essential to the success of the meeting, the happiness and proper entertainment of the delegates, to establish what practically amounted to a bar in a private room at the hotel, where the thirst of the delegates and visitors could be alleviated. The funds for this type of entertainment were obtained by private solicitation from those members of the Society who were sympathetic toward the supposed desires of the visitors for this kind of refreshment. It is our contention that this violation of the state and national laws relative to the dispensing of intoxicants added nothing whatever to the success of the meeting; that it decreased rather than increased the attendance at the business and scientific meetings; that it did not enhance the esthetic enjoyment of the entertainment offered the visitors; that it did no good to any one; and, if it did not do harm, it was at least a useless expense and a venture which is not calculated to elevate the medical profession in the eyes of the discriminating public. At the close of our session, the Association voted to hold the next meeting in Nogales, the inducement offered them being the ease with which the pleasure (?) of similar entertainments might be secured in the border city. No doubt, a very excellent scientific program will be presented and the chaotic conditions in the Santa Cruz County Medical Society may be stabilized by holding the 1931 session there, but these very laudable reasons for the invitation were not mentioned.

Concurrently with this convention in Phoe-

nix, the annual district meeting of the Lions' Clubs of Arizona was running in Mesa. The editor did not attend that entire meeting but was present at the Saturday afternoon business session, when the matter of the meeting place for 1931 was being discussed and voted upon. Douglas presented its plea for the meeting, with emphasis upon the advantages (?) of the Social Club in Agua Prieta. The other bidder for the meeting was Safford, whose delegates frankly stated they could offer no such inducements, but could properly house and feed the delegates while they performed the work of the convention. Although the Lions' Clubs of Arizona are composed of young business and professional men, the majority of whom are perfectly willing to drink when the occasion arises, their convention voted by a surprising majority to hold the 1931 assembly in Safford.

The comparison between the attitude of this convention of luncheon clubs and the convention of scientific, highly intellectual professional men,—as to what constitutes the proper function of a convention, was very illuminating and not a very comfortable illumination to a member of the latter group.

We are told that the International Rotary Convention, held a year or two ago in an eastern city, was regarded by the bell boys and attaches of the headquarters hotel as a failure. The complaint recounted to a delegate by the ticket agent in the hotel, in all seriousness, was that while the attendance at the convention was phenomenal, the side lines of merchandise offered by the bell boys and other hotel employees received so little patronage, that the convention was a failure to them. "They attended the convention sessions!" That was the remarkable complaint of this ticket agent.

In asking one of the delegates to a recent national medical convention, held in a southern city, how he enjoyed the meeting, he replied, "Not much, too much boozing and not enough attention to the meetings." Here, again, we are told that a free bar was installed in the hotel and any delegate could go there and drink anything from raw whiskey to beer, ad libitum. A high official of the Association is said to have been almost too far under the influence of liquor to deliver an important address. At many business conferences, officials were absent, either going under or coming out of, the influence of this hospitality. Can any one contend that this adds to the success of a convention to which delegates go a long distance for serious business? This particular Association is one of the great agencies for carrying on educational work for Good Health among school children! A moving picture of the inner circles of its annual convention might be a good thing to put out for this purpose.

Maybe the hot weather in Phoenix has gone to the editor's head, but at least this load is off his chest,—for the time being.

DR. LEROY JONES

The accidental death of Dr. LeRoy Jones, of Phoenix, brought to a tragic close a very promising career in the medical profession of Maricopa County. While Dr. Jones had only recently located in Phoenix, his quiet, courteous and affable manner had won friends wherever he made contacts. He came to Arizona the latter part of last year, passing the Board in January. After taking care of the practice of Dr. Charvoz for a month or two, he had just entered into association with Dr. Goodrich, when he was killed in an automobile accident on May 16th. He is susvived by his widow who was with him in the accident, but who was not seriously injured.

Dr. Jones was a graduate of Baylor University in 1928, taking special work in radiology and clinical laboratory before coming to Phoenix. He had expected to take up this class of work with the group of men associated with Dr. Goodrich.

DR. EDMISTON DARRAGH

Dr. Edmiston Darragh, of Bisbee, a member of the Cochise County Medical Society and the American Medical Associaton, and a practitioner for fourteen years in Bisbee, died there on April 21st, of nephritis. Dr. Darragh was born in 1872, graduated from the Northwestern University in 1902, coming to Arizona in 1916. He has been a member of the Calumet & Arizona Hospital staff during all of this period, except during the World War, when he was in service.

CANCER RESEARCH BY NEWSPAPER BALLYHOO.

For those who are directly acquainted by personal contact with the workers and by immediate observation of the methods employed, the experiments being conducted by Drs. Coffey and Humber, and their associates, in California, may be entirely scientific, ethical and accurate. But to the medical mind, attuned to other methods of research and more conservative routes of purveying information, the ballyhoo which is appearing in the newspapers and over the radio, is repellent. It will result in a loss of confidence which no results that may be obtained and no data accumulated will ever succeed in restoring. The eminence of these men in their profes-

sion, and the high esteem in which they are held by their confreres will not be sufficient to offset the shock to the conservative-minded medical reader when he sees such articles as the full page illustrated write-up which appeared in the Detroit Free Press of April 20, and perhaps in other syndicated newspapers. Aside from the distrust engendered by such announcements through the newspapers, the method of handling patients is not calculated to create confidence. In three or four centers in California, patients are received and given their injections, very much like wholesale vaccinations or antityphoid inoculations might be administered. From all information received, they are not kept under any kind of observation or control which will be of value in reporting results obtained. Much might be said on the proper method of conducting scientific experiments in cancer treatment. The proponents of this method insist that it is experimental; yet the methods of applying it are not those of scientific experiment, but of clinical use of a proven and assured treatment,—such as a vaccination against smallpox. We have been told that the first announcement of this method through the newspapers was very embarrassing to the doctors. It is even more certain that the continued newspaper and radio propaganda,—whether it can be controlled by the doctors interested or not,—is even more embarrassing to the entire medical profession.

In marked contrast to this technic of education is the conservative and dignified method adopted by the Garvan Cancer Research Laboratory in Baltimore, which is partly supported by The Chemical Foundation. It is certain that when the medical profession is presented with final results from these two sources, the latter will be received with confidence, while the former will require considerable salting to make it intellectually assimilable.

RADIO BROADCAST
(As Sponsored by the El Paso County Medical Society).

For twenty-five years laymen have been hearing that marvelously rapid strides have been made in the science and the practice of medicine. Yet, no organized effort has ever been made to supply the world with comprehensive information regarding medical discoveries and progress. We have been inclined to be shocked and surprised when our friends and former patients have had their backs rubbed because some glowing newspaper article had informed them that it was the cure for this, that and the other illness. Realizing the unfairness to the public, as well as the profession, the American Medical Association a few years ago instituted publicity programs of one sort or another to inform the public of what the medical profession had to offer. The late lamented president of the Texas State Medical Association, Joseph Dildy, adopted this publicity program, and centered his administration's activities around the annual health examination at his inauguration.

In harmony with the national and state program, the El Paso County Medical Soci-

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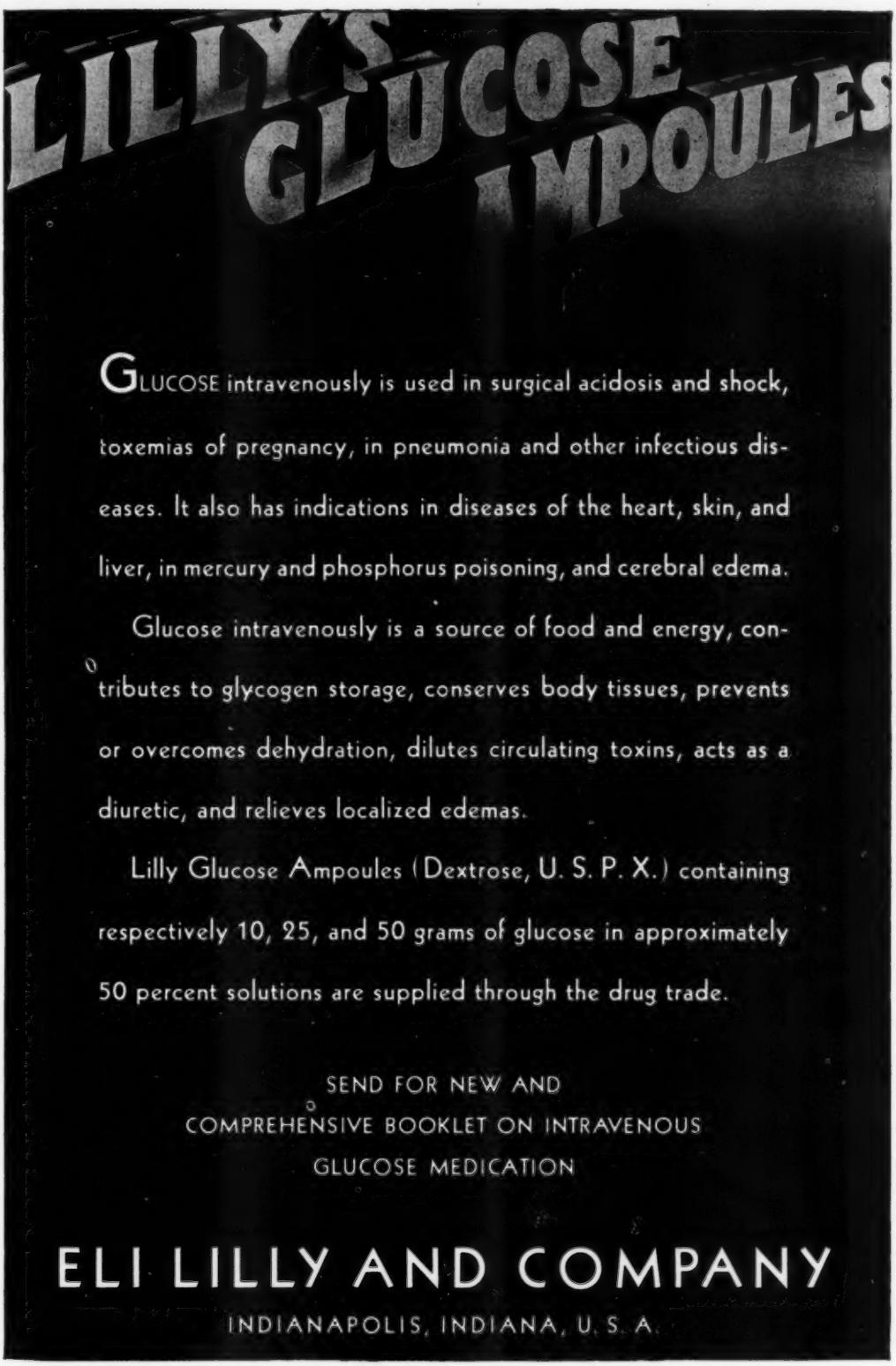
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ety decided upon a publicity campaign for a six months period, and starting in September they inaugurated a weekly radio broadcast over both of their local stations. Public health problems were featured over one station, and health conservation, with the broadcast being built around the annual health examination, on the other. There was a physician in charge of each station's broadcast, who secured the services of a speaker for each week over his respective station. No physician's name was used over these broadcasts. Doctors were known as Doctor W. D. A.H. or Doctor K.T.S.M. There were two ways of judging the effect of these broadcasts upon the public. First, a question box was kept at the station and those interested were asked to write in questions concerning anything of a medical nature. The other was that it seemed to be the indoor sport of many in the community to guess who the doctor was who was speaking over the station, and frequent phone calls would come in and with queries as to who the speaker was. From these it was concluded that the public was actually listening to the broadcast, and that they did so weekly, for frequently they would refer to statements made the previous week and sometimes for long periods back. Another accomplishment was, that all matters that pertained to medicine or health in any way that was presented the stations for broadcast were submitted to the committee to determine whether or not it was in any way offensive to regular medicine. In this way all irregular types of broadcasts were kept off the air, and El Paso has not been infested with this type of offensive information. These stations would naturally prefer to do business with the regular physicians of the city, and particularly when they were doing business with all the physicians in the city, rather than the isolated charlatan or irregular. This six months program cost the members of the society approximately six dollars apiece for the six months. And when it is considered that they are entirely amateurs at planning anything of a publicity nature, and particularly at radio broadcasting, they concluded that it was quite worth the time and expense incident to it, and there is little doubt that the society will continue the policy in the fall for another six months period.

Founded 1896 by Dr. Hubert Work

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New Equipment

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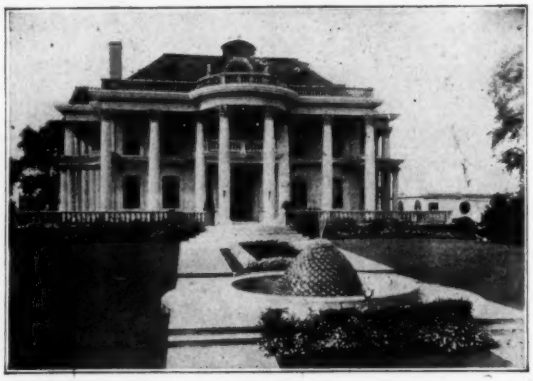
Superintendent



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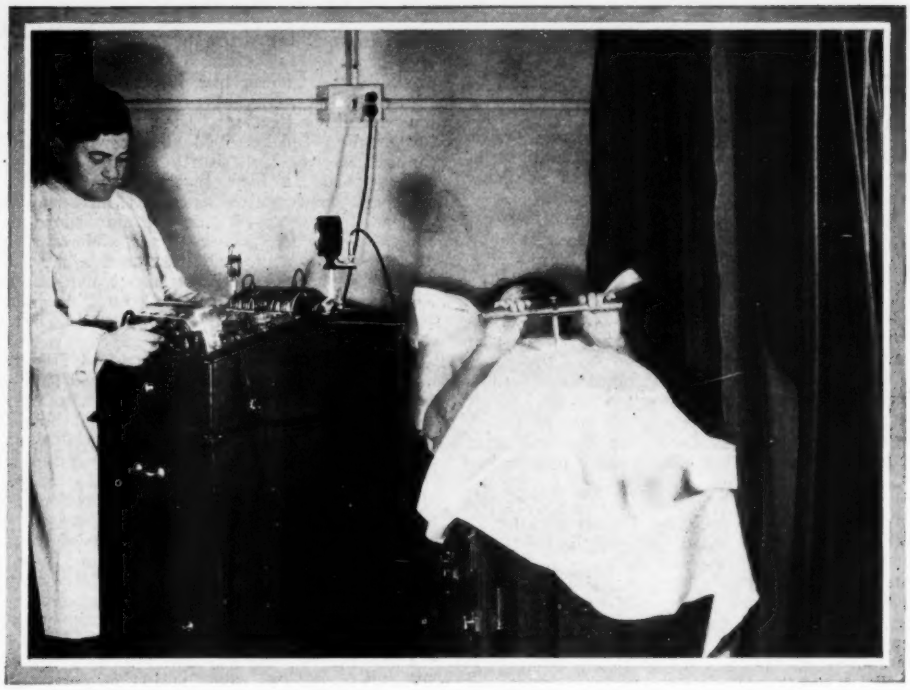
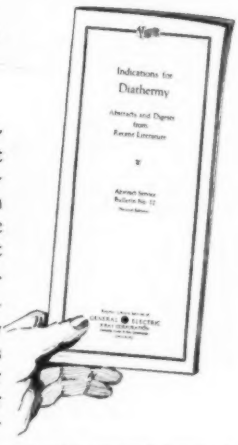


When is Diathermy of Value in Your Practice?



YOUR decision to use diathermy in the treatment of any condition will, of course, be based on recognized medical authority. Many physicians have become interested as a result of observing the many references to diathermy in current medical literature, and no doubt intend to investigate for themselves when opportunity presents. But a busy practice affords little of the time required in searching the files of the medical library, and it is put off indefinitely.

A preliminary survey of the articles on diathermy, published during the past year or so, is available to you in the form of a 64-page booklet entitled "Indications for Diathermy." In this booklet you will find over 250 abstracts and extracts from articles by American and foreign authorities, including references to more than a hundred conditions, in the treatment of which the use of diathermy is discussed.

If you number yourself among the physicians who have not adopted diathermy in practice, and desire to investigate this form of therapy in view of reaching your own conclusion as to its value in your practice, you will find this booklet a convenient reference. A copy will be sent on request.

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ARIZONA PUBLIC HEALTH ASSOCIATION ANNUAL MEETING.

The Third Annual Meeting of the Arizona Public Health Association was held April 22 and 23 at Phoenix, Arizona, with headquarters at the Adams Hotel.

The session the first morning was devoted to a general meeting of all sections, while the afternoon sessions and those of the following day were divided into three groups:—the Health Officers session, Water and Sewage session, and Dairy Inspectors session. Lantern slides and moving picture films presented at the meeting gave an added interest for the guests. An outstanding feature was the presence of the new Southern Pacific health car, which made its maiden trip for the benefit of those attending the meeting. This car was in charge of Dr. J. C. Geiger, Epidemiologist from the University of California. Dr. Geiger also gave a very interesting account of undulant fever.

There was a total registration of 117 guests for the two days. This list included Governor John C. Phillips, of Arizona, who gave an excellent talk on Health Policies at a dinner on the evening of the 22nd, and Dr. R. J. Stroud, State Superintendent of Public Health, who took a very active part in the meetings. Out-of-state visitors included the following representatives:—Dr. J. C. Anderson, State Superintendent of Health, State Board of Health, Texas; Chester Cohen, State Sanitary Engineer of Texas, and Dr. P. R. Outlaw, City Health Officer of El Paso. New Mexico was represented by Mr. Paul Fox, State Sanitary Engineer, who presented a paper on Disaster Relief. The United States Public Health Service was represented by Dr. H. A. Spencer, Acting Surgeon, San Pedro, California; Dr. F. T. Foard, Acting Assistant Surgeon, Stockton, California, and Mr. H. B. Hommon, Sanitary Engineer in Charge, San Francisco, California. Dr. J. H. Bushong, Asistant Secretary American Society Medical Milk Commissioners, Los Angeles, was also present at the meeting.

As a result of the interest taken in Dr. Harry Carson's talk on Incidence of Dysentery in the Salt River Valley, a stream pollutioin survey is being made of the Salt River from Granite Reef dam to the junction with the Gila. This work is being done in the co-operative arrangement with the United States Public Health Service and State Board of Health, and City Engineers' Department of Phoenix.

The new officers of the Association elected are as follows: President, Dr. H. T. Southworth, Prescott; vice-presidents, Dr. H. A. Reese, Yuma, and W. J. Jamieson, Phoenix; executive committee, Mrs Helene Thomas Bennett, Yuma; Dr. C. H. Laugharn, Clifton; T. P. Morgan, Phoenix, and M. R. Tillotson, Grand Canyon. Jane H. Rider, Tucson, was re-appointed Secretary-Treasurer.

EL PASO COUNTY MEDICAL SOCIETY
Regular Meeting, April 14, 1930.

Meeting called to order at 8 p. m., by the president, Dr. Paul Gallagher. There were 53 members and 2 visitors present.

Minutes of the previous meeting were read and approved.

The first number on the program was a clinical case presented by DR. GARRETT.

This was a Mexican man about 30 years of age, whose chief complaint was very severe upper abdominal pain radiating into back and chest. He was doubled up in agony. Had with him an x-ray film the report of which stated that there was probably present an obstruction of the cystic duct. However, on questioning him it was found that at age 18, he had a venereal sore and was given 18 intramuscular injections, then some drop medicine for awhile. Had received no other treatment as he thought he was well. Six years ago started having severe pains in legs, lasting one to eight hours. At first these pains came on at rare intervals, but as time elapsed they became more frequent. Then he started having upper abdominal pain at infrequent intervals—at first once a year, then ev-every few months, and now about once a week. The pain was severe enough to keep him from work. His family physician had four blood Wassermanns made, but as they were all negative, gave him no anti-leutic treatment. When Dr. Garrett examined him he found his pupils irregular and that they did not react to light. His knee reflexes, however, were still present. Cremasteric and superficial abdominal reflexes were absent; no ataxia as yet. He had a spinal fluid examination made and obtained the following report: Wassermann 1 plus, cell count 40, (mostly monos), globulin increased, colloidal gold showed a median zone reaction. Dr. Garrett considered this a typical early case of leutic infection of the central nervous system. In a patient such as this, the picture is perfectly clear when all the data is obtained, but in a hurried examination we are apt to jump at some conclusion wide of the mark. The prognosis in this particular case is not especially good, but the fact that as yet the patient has not a typical tabes is in his favor and with modern treatment the prognosis as to arrest should be favorable.

CAPT. PRATT discussed the case. It seemed to him that as the man walked the room, he showed a little difficulty in maintaining his equilibrium. It is not unusual fo: cases with abdominal crises of central nervous origin to fall into the hands of sur-

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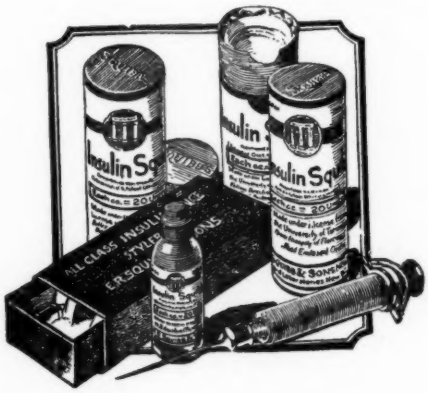

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geons. As regards treatment, the sooner this is instituted, the better the prognosis. If you push mercury and potassium iodide and especially tryparsamid and then follow with fever therapy (preferably malaria) and follow tha tagain with tryparsamid and mercury, you may expect results.

The next number on the program was "Some Interesting Lung Cases," by DRS. EGBERT, ARNOLD and GIBSON. These consisted of x-ray views of the chests of patients projected on the screen, together with a brief resume of the physical findings, history, course, etc. The first case was that of a young man with a rather typical case of pulmonary tuberculosis, who had received three doses of pneumothorax. The second case was another typical case of pulmonary tuberculosis, who had had a spontaneous pneumothorax associated with pneumonia in 1928. Since then thoracoplasty had been performed. An interesting feature of this case was that he had a calcified pleura, which is a rare bit of pathology. After the thoracoplasty, fragments of this calcified pleura dropped down in the lower part of the pleural cavity, as demonstrated by the x-ray. Another interesting feature of this case was the ingenious method of applying pressure to the operated side without producing pressure to the contralateral side. This method consisted of a sling or hammock in which the patient lay on his side and was suspended from pulleys to a frame work very much the same as is done in suspending a fractured leg. It had been devised by Dr. Arnold. The third case was that of a man, 66 years old, who had been sent here with a diagnosis of bronchiectasis. But Dr. Egbert was of the opinion that in addition to "bronchiectasis," the patient had pulmonary cancer. He said that cancer was often engrafted into a bronchietasis and that the latter is the commonest complication in cancer. The fourth and last case was that of an old man, past 60, who undoubtedly had an old tuberculosis, but in addition to this, Dr. Egbert suspected cancer, as the physical findings did not account for all his symptoms.

The next number on the program was "Focal Infection" by DR. L. A. NEIL, (dentist). Dr. Neil stated that "a better title would have been 'Some Improvement, Local and Systemic, Following Pyorrhea Treatment'" He also said that while a great deal of thought and study had been given to the abscessed tooth, but little has been said about the seriousness of pyorrheal infection. Forty or fifty inches of gum tissue gives quite a good-sized area for infection. A singled rooted tooth is much easier to free of infection than a double rooted one. The primary factor in all these cases of pyorrhea is traumatic occlusion. The treatment consists of correction of this traumatic occlusion and curetment. Successful treatment in pyorrhea should aim at the elimination of infection. He then showed a number of x-ray film taken before and after treatment. In all of them great improvement was noted. Also, the patient's general health had been materially benefitted.

DR. CATHCART, in discussing Dr. Neil's roentgenograms. said that in all of them the bone regeneration stood out very clearly.

DR. FELIX P. MILLER then read a preliminary infecting the pleura. To avoid this, many methods Pneumatic Bags. Drainage of an infected lung across an area where pleural adhesions are not complete entails danger of collapsing the lung and of infecting the pleura. To avoid this, many methods have been advocated to cause adhesions between the parietal and visceral layers of the pleura. In 1928, Dr. Miller began the use of rubber bags for this purpose. At first he used Barnes or Voorhees' bags and then the Pilcher prostatic rubber bags. He found them clumsy and adaptable for the conditions. So in 1929, he devised a rubber bag that is much more suitable for the purpose. He has found that external pneumolysis by means of these pneumatic bags will be of distinct value for safety, first, as a preliminary procedure to transpleural operation for the approach of tumors of the chest, and third, as a preliminary operation in causing compression of diseased conditions in the lung where it is not desirable to open the pleura.

Dr. Miller then presented the case of a patient who had had a lung abscess and had been treated by the use of these bags. He was entirely recovered in every way with the exception of a pleurobronchial fistula, which was still draining. This been expected.

DR. EGBERT thought the bag very interesting and that it undoubtedly had a purpose to serve. He asked Dr. Miller how long would the tissues bear the insult of pressure without necrosis. Dr. Miller stated that in the case presented, the bag had been left in two weeks, that whether or not necrosis occurred, depended on the amount of pressure used. On tuberculous processes, it would not be desirable to exert much pressure.

DR. R. B. HOMAN thought the bag should be very useful in cases of lung abscess. In the particular case presented, it had worked wonders.

DR. W. W. BRITTON then gave a case report of a patient who had come in the preceding Saturday and who brought his x-ray film and diagnosis with him. He was a man aged 21. On looking at the x-ray, Dr. Britton at first agreed with the diagnosis of spontaneous pneumothrax with effusion, but after making a physical examination, he could not. There had never been the distressed condition that should be present after a spontaneous pneumothorax and there was an absence of the tinkle sound that he has always found in these com-

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bined conditions. The fluid level showed no changes in change of posture.

DR. EGBERT stated that spontaneous pneumothorax in the lower lobe produced fluid in the pleural cavity almost immediately. In this case one might conceive of a partial pneumothorax.

DR. CATHCART said that the x-ray indicated to him simply encysted fluid.

The president announced that the personnel of the various committees for the meeting of the Medical and Surgical Association would be announced at the first meeting in September.

The application for membership of Dr. S. E. Wilson was then read. He was unanimously accepted into the society.

The president announced that the next meeting, that of April 28th, would be held at the Red Cross Building of William Beaumont Hospital, the program to be supplied by the staff members of that hospital.

There being no further business, the meeting adjourned at 10 p. m.

S. H. Newman, M. D.
Secretary-Treasurer.

DR. R. J. STROUD, of Tempe, State Health Officer for Arizona, left June 8th for eastern points. He will be gone about a month, during which time he will attend several medical conventions, including the American Public Health Association and the American Medical Association. To the latter convention he is the official member of the House of Delegates from Arizona.

DR. ORVILLE H. BROWN of Phoenix, attended the New Mexico Medical Society's meeting at Raton, June 4 to 6, as the fraternal delegate from Arizona. He presented a paper before the Society.

DR. A. M. TUTHILL, of Phoenix, after ten years retirement from the army, following his distinguished services during the World War, has resumed active service and has again taken charge of the brigade which he commanded during the war. He has been honored by having a new army post named for him, and is now on active duty with his brigade at "Fort Tuthill" in Colorado. His many professional friends will welcome the news that General Tuthill is again in physical shape for such duties.

DR. E. PAYNE PALMER, of Phoenix, will leave withn a few days for the Medical Association in Detroit, where he is a member of the corps of demonstrators on the treatment of fractures, in the Scientific Exhibit. Dr. Palmer will exhibit the treatment of fractures of the scapula.

DRS. ORVILLE H. BROWN and LOREN C. BARLOW, of Phoenix, have moved their offices from the Goodrich Building to the Security Building, No. 422, where they may hereafter be found.

DR. D. R. GASKINS, of Phoenix, has opened offices at 210 Security Building, where he will practice the specialties of diseases of the Ear, Nose and Throat.

OPENING AT WINSLOW, ARIZ.: Mrs. C. Prewitt Gregg, the widow of Dr. Gregg, who recently died suddenly while enroute to Los Angeles with a patient, announcse that there is an opening in Winslow for a physician. The purchase of the office equipment is all that is requested of any one taking over this locatoni.

DR. VERON KENNEDY, of Phoenx, has returned to the Mayo Clinic, Rochester, Minn., for further treatment of duodenal ulcer.

DR. HOWELL RANDOLPH, of Phoenix, is spending a month in the Mogollon Mountains, combining a vacation with duties as physician for the Boy Scouts' Camp near Payson.

MONOGRAPH ON MALARIA. By D. Drysdale Anderson, M.R.C.S., L.R.C.P., Medical Officer of Health, West African Medical Staff. Published by S. J. Pridgen Co., Atlanta, Ga. 28 colored plates and 47 drawings. $6.00.

This is the initial volume of the Ready Reference Medicine and Surgery, to be issued in monographs by these publishers. The introduction to this volume is written by Dr. Allen Bunce of Atlanta. There are several unique features in this monograph, which will be carried through other volumes. In this work, the author covers the subject in a very concise way which the busy practitioner will appreciate. There is a very large marginal space for notations by the reader. There are perforated blank pages in the back of the volume for tipping in future additions. There are also blank pages following the perforated sheets, so that personal memoranda of cases may be written in by the doctor. The colored illustrations, and the black and white drawings are very good.

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SHORT POSTGRADUATE COURSES

The medical men in such districts as the southwest will welcome the tendency of medical schools to give short, intensive, courses on selected phases of clinical medicine. Several of the schools offer such opportunities this year. One of the most attractive is the Symposium on Kidney in Health and Disease, to be held at the University of Minnesota Medical School, Minneapolis, July 7 to 18. The University offers this ten day course free of charge. With forenoon and afternoon lectures and clinics by eminent clinicians, including Prof. F. Volhard, Marshall, Rowntree, Keith, and others, such a course as this offers an excellent opportunity to any one who wishes to bring his knowledge of kidney physiology, pathology and clinical disease up to date.

DR. CHARLES C. TIFFIN, of Seattle, who was on the program of the Arizona State Medical Association meeting in Phoenix, in April, was detained at the last minute by an emergency operation upon his daughter for acute appendicitis. He has sent his regrets to several friends in Arizona, who have transmitted them to this journal for publication. The last reports received were that the daughter was practically out of danger, which the doctor's friends in Arizona will be very glad to hear.